THE JOURNAL OF PERIODONTOLOGY

October
1955

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 26
NUMBER 4

CONTENTS

Periodontal Case Management in Hemorrhagic Disease	247	John F. Prichard
Periodontal Osseous Surgery: Osteoplasty and Osteoectomy	257	Nathan Friedman
Experimental Occlusal Trauma	270	S. N. Bhaskar, et. al.
Healing of Naturally Occurring Periodontal Pockets about Vital Human Teeth	285	Melvin L. Morris
The Healing of Periodontal Wounds in Dogs	292	M. T. Jansen, et. al.
A Quantitative Evaluation of the Effect of Copper on Alveolar Bone Loss in the Syrian Hamster	301	Emmett R. Costich
Case Report: Successful Handling of Gingival Enlargement of Complex Etiology	306	Edward Bressman
The Nitrogen and Lipid Content of Compact Bone from Guinea-Pig Mandible	309	Philip Person, et. al.
Editorial	314	
1955 Diplomates of the American Board of Periodontology	319	
Announcements	313, 315-320	

Publication in the JOURNAL OF PERIODONTOLOGY *is not to be understood as endorsement by the Journal or the* AMERICAN ACADEMY OF PERIODONTOLOGY *of opinions expressed in any article, abstract or extract. The object of publishing the various views is to present them without prejudice, to the reader for his own evaluation.*

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Thirty-ninth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Sheraton-Palace Hotel, October 1955

San Francisco, California

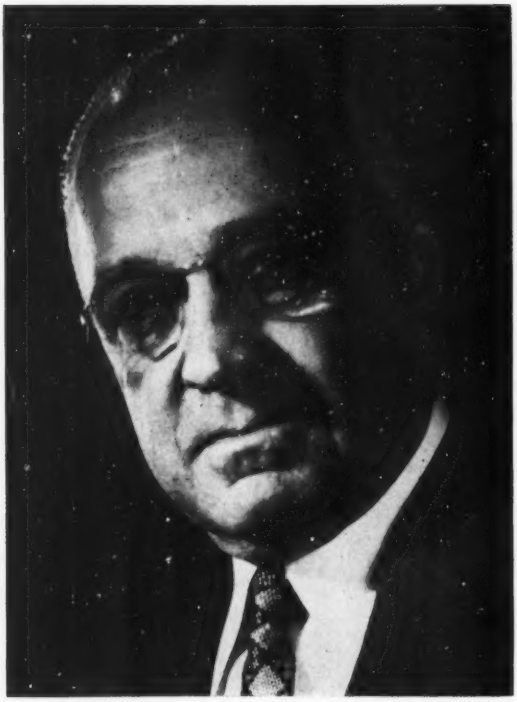
1956 Spring Conference

of

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Ann Arbor, Michigan

April 30, May 1, May 2



CLARKE E. CHAMBERLAIN, D.D.S., F.A.C.D.

Secretary of the American Academy of Periodontology

One of Dr. Clarke Chamberlain's duties as Secretary of the American Academy of Periodontology is to care for an infinite number of duties associated with the annual meeting. All those who have attended these meetings recently are amazed at their smooth operation, and to Dr. Chamberlain goes most of the credit.

Dr. Clarke Chamberlain is well known to all periodontists because of his activities in the Academy and on the Board of Periodontology. He has been on numerous committees, held many offices in dental organizations, and is active in community affairs since he belongs to the Royal Order of Jesters, Masonic Lodge and Rotary Club.

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY-FIVE

Periodontal Case Management in Hemorrhagic Disease

by JOHN F. PRICHARD,* D.D.S., *Fort Worth, Texas*

THERE have been many articles published on blood dyscrasias, particularly the leukemias, calling attention to the characteristic diagnostic signs and symptoms, the importance of early recognition and the grave consequences of dental treatment, especially of tooth extraction. In 1949 Wentz[1] described the gingival picture in acute leukemia as one of necrosis, ulceration, spontaneous hemorrhage and hyperplasia.

It is the purpose of this report to discuss clinical management of periodontal disorders in hemorrhagic disease. Oral bleeding in these patients is a serious matter which requires control for comfort and sometimes for the preservation of life itself. In fact, hemorrhage from the mouth may be the first symptom and the chief complaint.

Therapeutic objectives for many of these patients must be lowered considerably from our usual standards of success. Prognosis in these cases is sometimes grave and it is the aim of therapy to provide comfort, to prevent a too rapid regression, and for the most part, to maintain *status quo* in oral health—to give ground as slowly as possible. This provides a challenge to the best resources of the periodontist.

Bleeding abnormalities complicate the lives of many people. This is important to dentistry because hemorrhage from the gingiva or infiltration of oral tissue may be the first clinical manifestation of systemic disease. Many diseases of the blood and blood-forming tissues affect the periodontal structures and their correct management requires both precise diagnosis and close cooperation between dentist and physician.

In some of these conditions medical treatment can prepare the individual for the usual periodontal procedures in a normal manner, but in others periodontal treatment must be modified to be compatible with the systemic condition. In these cases, what we consider an acceptable periodontal result is also altered to conform to the limitation imposed upon treatment by the systemic disease. In cases of serious blood disorders the result is considered satisfactory if the periodontal condition can be improved enough to prevent unprovoked oral hemorrhage.

*Clinician in Periodontia Department, Baylor University, College of Dentistry.

This report is not being rendered from the perspective of the hemotologist, but of the periodontist. Therefore, none of the many classifications of blood dyscrasias is being offered. These are available in any standard work.

STUDY OF THE PATIENT WITH A HISTORY OF ABNORMAL BLEEDING

An individual with periodontal disease giving a history indicating a hemorrhagic disorder should have a blood examination including the following tests: coagulation time, clot retraction, prothrombin time, bleeding time and the platelet count. A tourniquet test and a complete blood count should be made.[2]

Coagulation time determines the ability of the blood to clot after it has been removed from the body. Bleeding time is a measure of capillary retraction as well as of the capacity of the blood to clot with the aid of tissue fluid since the test is based on the effect of a skin puncture in producing bleeding. False normal results may be obtained unless the tests are carefully done. Prolonged prothrombin time indicates vitamin K deficiency or an excess of dicoumarol in the blood. Prothrombinopenia from lack of vitamin K may be due to dietary deficiency or faulty absorption caused by the lack of bile salts such as occurs in obstructive jaundice, billary fistula and sprue, or in cases of liver damage. Prolonged use of acetylsalicylic acid may decrease the prothrombin. The administration of vitamin K usually corrects such cases but may not be effective in cases of liver necrosis. Quick states that the degree of reduction of the prothrombin level is a far better indicator of the bleeding tendency than the clotting time.[3]

Clot retraction depends chiefly on an adequate supply of normal platelets and delayed retraction suggests thrombocytopenia. Bleeding may occur when the platelets number as few as 100,000 and is usually profuse if the platelets are fewer than 50,000 per cubic millimeter. A positive tourniquet test indicates that the capillaries cannot stand increased pressure. This may be due to avitaminosis C, a low platelet count or to endothelial damage by infection or allergy. Calcium deficiency does not cause bleeding because calcium levels sufficiently low to interfere with coagulation are far below those compatible with life.

Abnormal bleeding may result from anatomical or physiological defects of the blood vessels or from defective factors in the blood itself. It may occur as a secondary manifestation of anemia or other diseases such as liver dysfunction, hypertension, renal disease or due to the use of anticoagulant drugs in medical treatment. There are many instances of pathologic bleeding in which the results of all hemostatic tests are within normal limits.[4] It is also worth emphasizing that *some* tests are normal in all these states. Thus, the bleeding time is perfectly normal in "bleeders disease" (hemophilia). Recognizing the unusual is not difficult when symptoms are alarming or marked. The real pitfalls for the dentist lie in those blood diseases in which dental and oral symptoms and complaints mimic well known types of dental disease.

HEMORRHAGE DUE TO DEFECTS IN THE BLOOD

Hemophilia: Hemophilia is an hereditary hemorrhagic disease in which the bleeding time is normal while the coagulation time is greatly prolonged. All other blood tests are usually normal except a prothrombin consumption test developed by Quick. Purpura does not occur and blood elements are normal unless altered by recent bleeding. The bleeding of hemophilia is deep in contrast to the hemorrhage of purpura which appears to be superficial as it can be seen in the skin and mucosa. At autopsy it is found in the deeper tissues as well.[5]

Hemophilia is called the most hereditary of all diseases and has been considered an exclusive disease of the male, being transmitted by the female. The hereditary pattern of hemophilia is being re-studied and

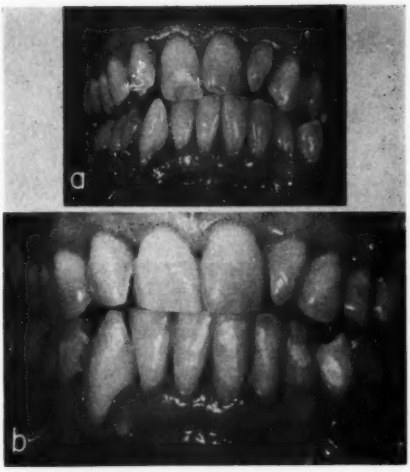


Fig. 1. Mrs. T. L., a 37 year old woman, with intermittent oral hemorrhage due to polycythemia vera: A. Clinical appearance with unprovoked hemorrhage from the gingiva of the right mandibular cuspid. B. Appearance of gingiva 18 months after treatment with tannic acid packs to prevent hemorrhage from scaling and instruction in home care of the mouth.

several investigators have obtained evidence that hemophilia does occur in the female although it may be less severe than that affecting the male.[6, 7]

CASE REPORT

F. H., a seventeen year old boy, was admitted to the City-County Hospital because of gingival bleeding. He was a known hemophiliac, having been admitted numerous times. He had been doing well since his last admittance except for "bleeding from his gums" which had become progressively worse.

Dental examination revealed a marginal gingivitis with heavy accumulations of materia alba and subgingival deposits of calculcus. He had a full complement of teeth except for the third molars.

At the time of his admittance his blood picture was as follows:

RBC 4,520,000	Stab. 4
Hbg. 14 gms, per 100 cc.	Seg. 64
WBC 8,200	Lym. 32
Bleeding time 1 min. 10 sec.	Prothrombin Time:
Coagulation time 19 min.	Patient 60 sec.
Type A RH positive	Normal 45 sec.
	76% of normal

Daily plasma units were ordered by the medical department with transfusions of whole blood as indicated to reduce the coagulation time to normal so that oral prophylactic measures could be instituted.

On the fifth day after admittance the coagulation time was 5 minutes and he was sent to the dental department for treatment. Thorough subgingival scaling was done with normal bleeding and there was no intermediate or secondary hemorrhage after he returned to the ward.

He was dismissed after fifteen days of hospitalization, having had four transfusions of whole blood and two daily units of plasma. He has had numerous admittances since then but no further oral bleeding.

Polycythemia Vera (Erythremia): Polycythemia vera or erythremia is a chronic progressive disease characterized by an increase in the number of erythrocytes, the amount of hemoglobin and the total blood volume.[8] The outstanding clinical feature is the dusky, brick-red color of the skin and mucous membranes. Bleeding and coagulation times are normal, the sedimentation rate is decreased and clot retraction is poor even though the platelet count may be greatly elevated, because the red cell mass is so great. The capillaries are diffusely dilated and there is increased capillary pressure which increases the hemorrhagic tendency.[9]

CASE REPORT

Mrs. T. L., a thirty-seven year old woman, was referred for periodontal treatment because of gingival bleeding. A diagnosis of polycythemia vera had been made four years before and she had been under medical supervision since that time. The referral was made with instructions not to use a scaler because she had bled constantly for seven days following a recent scaling treatment.

At the time of presentation her blood count was as follows:

RBC 6,600,000, WBC 7,120, Hbg. 13.5 gms.
Platelets 1,860,000 per cu. mm.
MCH 20.4
Differential count—PMN 88% (Myel. 3%, Juv. 3%, Stab. 15%, Mat. 67%)
EOS 7%, BASO 1%, Lym. 4
5 normoblasts per 100 wbc.

Figure 1A shows the gingiva as it appeared with spontaneous hemorrhage from the marginal gingiva of the lower right cuspid. A probe was not used but there was a marginal periodontitis,[10] with bone resorption evident in the roentgenograms. Various

Fig. 2. Mr. E. A., a 58 year old man, with undiagnosed thrombocytopenic purpura: A. Appearance before treatment. The maxillary gingiva has a peculiar mottled appearance. B. Five days after upper gingivectomy with extensive ecchymoses. C. Appearance three months after treatment of the mandibular teeth with 10% paraformaldehyde dressing and subgingival scaling. A physical reevaluation had revealed a previously undiagnosed thrombocytopenic purpura.

methods of management were attempted, using the left maxillary posterior teeth as a trial area.

Following the technic advocated by Vastine,[11] tincture of metaphen was pumped into the gingival sulci with a rubber cup for three successive days to reduce the bacterial count before occluding the pockets. Periodontal surgical dressing with 10 per cent paraformaldehyde[12] for chemical gingivectomy was then packed into the pockets to reduce pocket depth and expose subgingival deposits of calculus. Neither objective was obtained as the pocket wall was not adequately reduced and severe hemorrhage occurred because of the sloughing of necrotic tissue.

Cotton ropes, saturated with various drugs, were packed into the pockets before and after scaling. Epinepherine 1 to 1000, 8 per cent zinc chloride and Monsel's Solution all failed to prevent or control hemorrhage. Scaling was finally accomplished by leaving packs of a saturated solution of tannic acid in place for twenty-four hours before treatment and then immediately repacking for twenty-four hours. There was very little hemorrhage during instrumentation and only minor post treatment bleeding. The packs were painful and the gingiva healed very slowly because of the sluggish circulation and dilated blood vessels.

Instruction in oral hygiene was initiated by having the patient gently massage the gingiva with large cotton balls. Later, a soft tooth brush and a flexible rubber point were used. The mouth has been maintained in this manner for two years and while the case does not conform to our usual criteria of success, our basic objectives were attained. Gingival bleeding has been controlled for the first time. Attention should be called to the fact that rational therapy (subgingival curettage, home care, etc.) was made possible through the use of adjunctive methods mentioned. The patient reported that for the first time in four years she does not have the taste of blood in her mouth. Figure 1B shows the oral condition after treatment.

Purpura Hemorrhagica: Purpura is but one manifestation of abnormal bleeding. The term refers to extravasations into the skin or mucous membrane with their deep red or purple color. They may appear in the form of petechial showers or as large ecchymotic areas.

Primary thrombocytopenic purpura is a hemorrhagic disease of unknown etiology and variable course associated with a reduction in the platelet count, prolonged bleeding time, diminished clot retraction and increased capillary fragility with normal coagulation time and normal prothrombin concentration.

Secondary thrombocytopenic purpura occurs when there is a reduction in the platelet count due to a known etiologic agent such as drugs, chemicals, radioactive substances, acute infection, allergies, etc.

CASE REPORT

Mr. E. J., a fifty-eight year old man, was referred by his dentist for periodontal treatment. Most of his posterior teeth had been extracted, one or two at a time, during the past fifteen years. Oral roentgenographic examination revealed a marginal periodontitis with moderate bone resorption. Periodontal pockets averaged from four to six millimeters in depth around the remaining teeth. The gingival condition is shown in Figure 2A.

The patient stated that he was a "bleeder" and had always hemorrhaged freely from wounds. He was being treated by a physician for an enlarged heart and vitamin K had been prescribed because of the hemorrhagic tendency. Ecchymotic areas had not been observed on his skin and he had never bled spontaneously.

All of his extractions had been done by the same dentist who stated that the sockets had been packed following extractions but that no other treatment had been necessary. Petechiae were not present in his mouth while under my care. Subgingival scaling did not cause excessive hemorrhage and was not followed by secondary bleeding.

A gingivectomy was done on the maxillary anterior teeth. The wound bled freely during the operation but was not a problem until the surgical dressing was applied. The soft dressing was lifted out of place by the flow of blood but by constantly readapting and holding it under pressure the bleeding was controlled. When the material became hard enough to remain in position there was no further bleeding. No post operative hemorrhage occurred, but when the dressing was removed on the fifth day an ecchymotic area extended completely around the wound as shown in Figure 2B.

The patient was referred to a hematologist for a complete blood study. His findings were as follows:

Erythrocytes 5,100,000 per cu. mm.

Leukocytes 9,600

Hemoglobin 89%—13.75 gm. per 100 cc.

Bleeding time—stopped with pressure after 15 minutes

Coagulation time—4 minutes

Platelets 40,000 per cu. mm.

Prothrombin time:

Clotting time of control plasma—13.5 seconds
Clotting time of patient's plasma—15 seconds
Per cent of normal prothrombin time—76%

Prothrombin Consumption Test:

Clotting time of control serum:
15 minutes after clot formation—9 seconds
1 hour after clot formation—66 seconds
Clotting time for patient's serum:
15 minutes after clot formation—9 seconds
1 hour after clot formation—12 seconds

Tourniquet Test: Strongly Positive

Clot retraction incomplete after 24 hours

Diagnosis: Thrombocytopenic purpura

The chest examination revealed no abnormalities and splenectomy was recommended but the patient refused to have the operation.

The maxillary gingiva healed slowly but when complete it had acceptable physiologic form and the pockets were eliminated. The mandibular teeth were treated by subgingival curettage and periodontal surgical dressing with 10% paraformaldehyde was used to reduce pocket depth. The result is seen in Figure 2C, and is not as satisfactory as that obtained in the maxilla but there was no abnormal bleeding. This case emphasizes the importance of a complete study of any patient with a history of abnormal bleeding preoperatively.

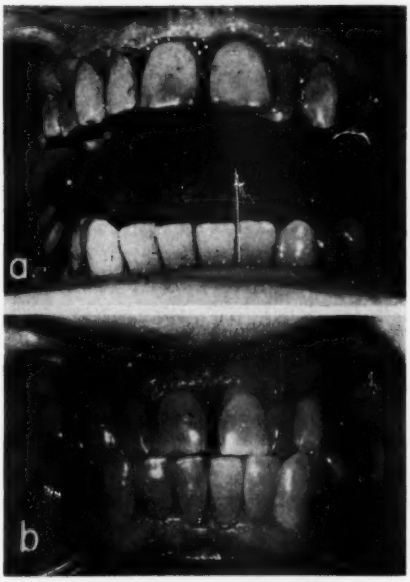


Fig. 3. A. Mrs. C., a 70 year old woman, as examined for control of unprovoked gingival hemorrhage associated with hereditary hemorrhagic diathesis. B. Mrs. C. two years after treatment with subgingival packs, scaling and improved home care. The architectural form is poor, but the tissue is firm and there has been no recurrence of the bleeding.

Hereditary Hemorrhagic Diathesis: There is a group of atypical hemorrhagic disorders affecting both sexes with non-specific and inconsistent abnormalities in the hemostatic tests. Bleeding from the nose and mucous membranes is common but purpura is rare, the bleeding time is usually prolonged, clot retraction is often normal, hemarthroses are uncommon, and a positive family history is often obtained.[13] Many titles have been employed for entities within this group, the most common one being pseudo-hemophilia.

CASE REPORT

Mrs. C., a seventy year old woman, was referred by her physician for control of oral hemorrhage. She had been bleeding intermittently, especially at night, for several weeks. He had prescribed vitamins C and K without benefit. Her blood pressure was 180/100, weight 192½ pounds and she had a low red cell count which was due to secondary anemia. Her bleeding and clotting times were both within normal limits. Her mother was a bleeder. She had

two grown children: a normal daughter and a son, a bleeder, whose history is included in this report. Her history included several instances of gastro intestinal hemorrhage and in January, 1944, her abdomen was explored for "unexplained bleeding from the upper gastro intestinal tract." No ulcers or other abnormalities were found. The gallbladder, which was inflammed and enlarged was removed, also the appendix. She has not had intestinal hemorrhages since the operation, but has vomited blood a few times. Abdominal surgery is known to frequently reduce the bleeding time and to increase capillary resistance.[14]

Figure 3A shows the oral condition at the time of our examination. There was hemorrhage from the gingiva of the maxillary anterior teeth and the right mandibular posterior teeth. The clots were removed and the hemorrhage was controlled by placing peridontal surgical dressing over the gingiva. Subsequent treatment consisted of subgingival scaling and surgical dressing was applied after each treatment. Excellent cooperation has been obtained in home care of the mouth and there has been no gingival bleeding for the past eighteen months. Figure 3B shows the mouth after treatment, and though it leaves much to be desired, it may be accepted as a satisfactory result in this case.

Mrs. C's son, a forty year old man, had postponed dental treatment because of fear of bleeding. His history revealed that in October, 1942, bleeding and coagulation tests were made in preparation for a third molar extraction. The clotting time was 7 minutes, 30 seconds (capillary pipette method) and the bleeding time was 35 minutes. Additional blood studies were then made as follows:

RBC 4,900,000 per cubic mm.

WBC 8,500 per cubic mm.

Hbg. 105%, 14.4 gms. per 100 cc.

Platelet count 140,000

Prothrombin index 1 (normal 1)

Vitamin C 1. 02 mg. per 100 cc. of plasma (normal Vitamin C 0.7 to 1.3 mg. per 100 cc.)

Blood Bromides 55.5 mg. per 100 cc. of serum (normal—0.8 to 1-5 mg. per 100 cc.)

Wassermann, Kline and Kahn Tests were negative.

In June, 1943, following eight months of liver, vitamin and calcium therapy, the bleeding time was 3 minutes and the coagulation time was 3 minutes, with a platelet count of 200,000. The third molar was removed and the post-operative hemorrhage was so severe that he was hospitalized for 72 hours. His last blood study was made in July, 1948, and was normal except for a platelet count of 120,000.

Oral examination revealed firm gingiva with shallow pockets and heavy deposits of subgingival calculus. There was very little hemorrhage from probing. The following blood tests were made:

RBC 4,910,000 Hbg. 14.0 gms.

WBC 10,500 Color Index 0.85

Platelet count 212,000 per cubic mm.

Coagulation time 5 minutes, 25 seconds

Bleeding time 2 minutes

Prothrombin time: patient: 17 seconds
control: 15 seconds

Clot retraction complete within 3 hours

Tourniquet test: negative; no evidence of increased capillary fragility.

His periodontal condition required only the removal of subgingival deposits and instruction in home care of the mouth. Three quadrants were treated by subgingival scaling with no bleeding but following the fourth and final treatment he bled for fifteen hours. The patient could not call for professional aid because he was in an isolated area at the time.

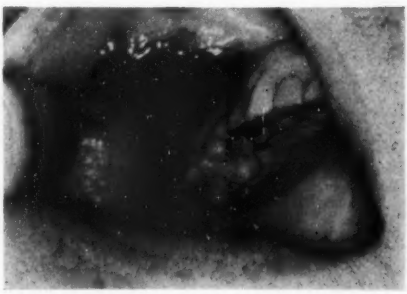


Fig. 4. A congenital nevus venosus or cavernous hemangioma of the buccal mucosa.

BLEEDING DUE TO ANOMALIES OR ABNORMALITIES OF THE BLOOD VESSELS

Hemorrhage may occur because of tumors or anomalies of blood vessels within the oral cavity. Figure 4 shows a congenital nevus venosus or cavernous hemangioma of the buccal mucosa.[15] This is a tumor composed of large irregular blood vessels, dark red, almost purple, in color and it does not blanch under pressure. It extends from the cheek across the mucoalveolar sulcus of the maxilla to the attached gingiva; it is bulky and would be liable to injury during any dental procedure. The tumor has remained constant in size, which is characteristic of its congenital origin. Histologically, the accumu-

lation of vessels in a hemangioma is not demarcated by a fibrous capsule and it is supplied by its own artery and vein.[16] Thoma,[17] refers to a case of fatal hemorrhage from a cavernous hemangioma following tooth extraction.

Burford, Ackerman, and Robinson[18] recommend treatment of cavernous hemangiomas in the oral cavity of adults by injection of a sclerosing solution, sodium morrhuate, as it is much simpler than surgery and radiation therapy is not indicated as they are relatively radioresistant.

A comparatively rare vascular anomaly known as Hereditary Hemorrhagic Telangiectasis is characterized by multiple dilation of capillaries and venules in the skin and mucous membrane. Hemorrhage may occur spontaneously or as a result of friction or trauma the bleeding is always on the surface, never interstitially. Electrocoagulation can be used to destroy lesions on the skin and in the oral cavity, but in some cases new telangietases appear as rapidly as the old ones are destroyed. Figure 5 shows the vascular anomaly as a round elevated spot on the tongue.

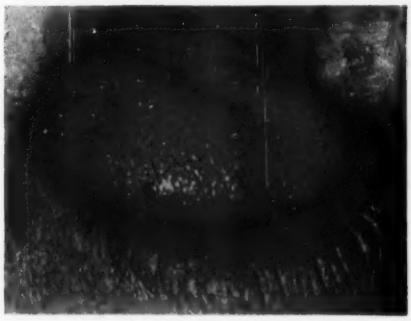


Fig. 5. The vascular anomaly appears on the tongue as a round elevated bright red spot. The telangiectasia is familial.

Some skin diseases have oral lesions that hemorrhage spontaneously. Epidermolysis bullosa is a rare skin condition of hereditary origin in which bullae arise on pressure and from traumatism.[19] They may be hemorrhagic.[20] The etiology of the disease is unknown and the pathology is not specific although in most cases there is a diminution or absence of elastic tissue in the corium. Figure 6A shows the oral lesion on the gingiva, tongue and buccal mucosa and 6B illustrates the typical skin lesion.

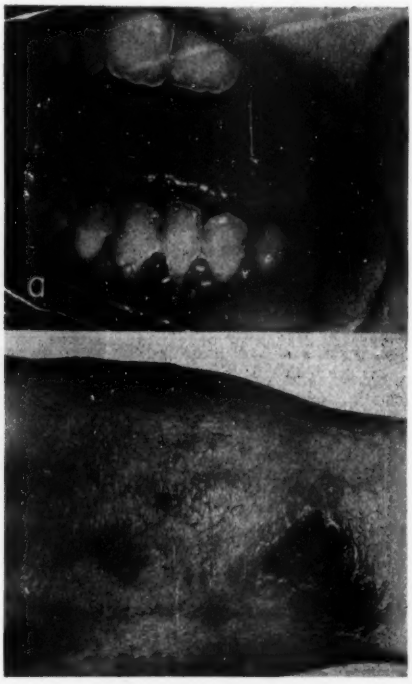


Fig. 6. A ten year old boy with epidermolysis bullosa: A. Illustrates the oral lesion on the gingiva, tongue, and buccal mucosa, the lips are inelastic. B. Illustrates the skin lesion.

SCORBUTIC HEMORRHAGE

Capillary bleeding occurs in scurvy, usually at points of mild trauma and sites of stress or where the hydrostatic pressure of the capillaries is elevated. Oral lesions do not appear in the edentulous mouth.[21] Plasma ascorbic acid levels are not diagnostic of scurvy but indicate only recent intake of vitamin C. The ascorbic acid

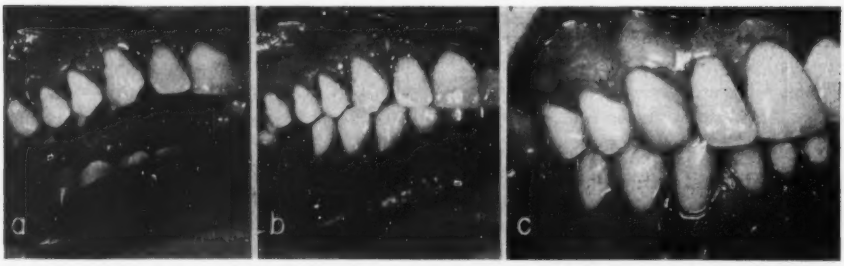


Fig. 7. A 29 year old woman whose chief complaint was pain and hemorrhage during mastication. A. The gingiva before treatment. B. After removal of gross deposits, 30 days of ascorbic acid therapy (400 mg. daily) and use of a soft tooth brush. Edema and inflammation are reduced, but improvement is no more than would be expected following removal of gross deposits. C. Four years after treatment. The gingival architecture is acceptable and there has been no recurrence of bleeding.

level in the white cell platelet layer of centrifuged blood is more significant and levels below 0.1 mg. per 100 cc. have a close correlation with the presence of scurvy.

Adult scurvy is infrequent today except among the indigent, the insane, the alcoholic and the food faddist.[22] Relative deficiency states of ascorbic acid are more common and until the human study by Crandon et al[23] gingival and oral lesions were believed to represent an early manifestation of this deficiency. The periodontium of their human experimental subjects did not show changes until long after the general symptoms of scurvy appeared.

Burket states that while vitamin C is necessary for wound healing in any tissue, the clinician can expect little result from the administration of this substance in the hope of eliminating "bleeding gums."[24]

CASE REPORT

Miss A. L., a twenty-nine year old woman with chief complaint of "sore bleeding gums." There was a marginal periodontitis with inflammation of the marginal and attached gingiva, a suppurative exudate, and tumefaction of the papillae. Subgingival deposits were visible under the retracted gingival margins and were black due to incorporation of blood elements. Figure 7A shows the gingival disease.

Her general appearance was poor, her skin was mottled, and she seemed mentally sluggish. Her weight was 117½ pounds, temperature 98.6, pulse 60, and blood pressure 110/80. She had not had chills or fever.

A preliminary scaling was done to remove gross deposits, and she was referred to her physician for physical evaluation. The following report was received:

RBC 4,800,000, WBC 8,700, Hbg. 92%

Differential count: eosinophilis 6, basophile 1

Staff 1, segmented 46, lymphocytes 42

monocytes 4

vitamin C level of the serum 0.25 mg. per 100 cc. (normal, .7 to 1.3)

calcium 9, 1 mg. per 100 cc. of serum (normal 9.0 to 11.0).

ascorbic acid 100 mg. four times daily had been prescribed.

In order to evaluate the vitamin therapy no further local treatment was done. Thirty days later the papillae were less edematous and the redness was reduced as shown in Figure 7B, but the gingivitis persisted. Improvement was no more than would be expected from removal of the gross deposits and instruction in oral hygiene as she could use a soft tooth brush after the heavy deposits were removed.

The ascorbic acid was discontinued and periodontal treatment including gingivectomy was done. The results as seen in Figure 7C shows the firm, pink, gingiva four years after treatment with an acceptable architectural form as maintained by the patient's home care and oral prophylaxis.

There may be hemorrhage from the gingiva in chronic gingivitis due to vascular changes, ulceration of epithelium of the sulcus or macerated areas of tissue. Unprovoked bleeding may occur in necrotizing ulcerative gingivitis as the result of exposure of the enlarged connective tissue by desquamation of the necrotic surface epithelium.[25] It should be remembered that necrotic lesions frequently occur in blood

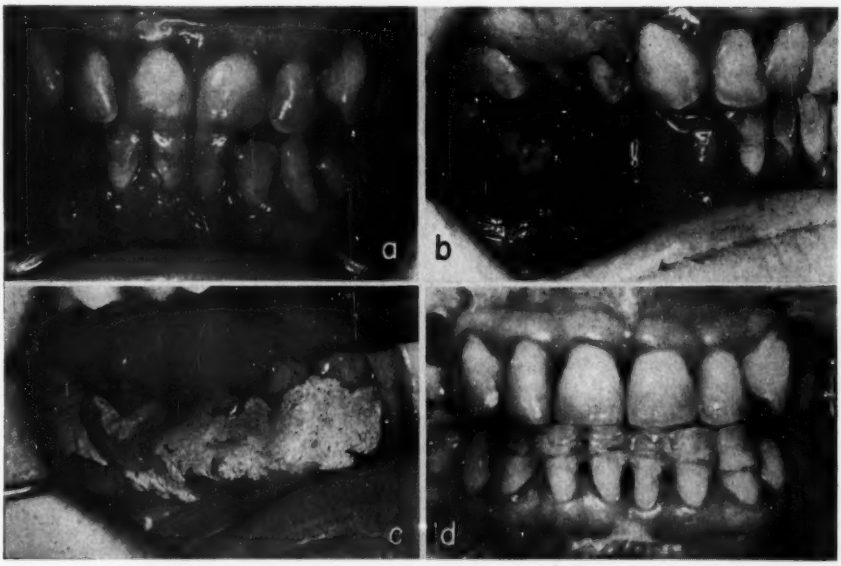


Fig. 8. A. & B. Mrs. S. as examined for control of hemorrhage 72 hours after her dentist had scaled the right mandibular quadrant. C. After hemorrhage was controlled by subgingival packs saturated with tannic acid solution and covered with periodontal surgical dressing. D. After gingivoplasty the gingiva is firm and pink, but the margins are thick and architectural form is poor. The mandibular incisors are ligated with stainless steel wire and acrylic for temporary stabilization.

dyscrasias, especially agranulocytosis, and accurate diagnosis must be made.[26,27] Bacterial smears are of little value since it is known that the microorganisms responsible for the characteristic symptoms of necrotizing ulcerative gingivitis occur widely if not universally in mouths.

The gingival blood vessels are contained in papillary connective tissue projections between the pegs of the overlying epithelium. Normally stratified squamous epithelium with a hornified surface layer protects the connective tissue and blood vessels from injury.[28] In inflammation the epithelium is thinned, there is no hornified layer and varying degrees of degeneration may be present. The combination of dilated capillaries, increased capillary permeability and reduced protective covering all contribute to the ease with which gingival bleeding may occur.

Because of these vascular and tissue changes, prolonged gingival hemorrhage may follow instrumentation for the removal of subgingival deposits.

CASE REPORT

Mrs. S. was referred to our office because of gingival hemorrhage of three days duration following subgingival scaling of the right mandibular posterior teeth. She had been treated by three dentists and a physician; vitamin K had been injected and pressure packs applied locally without success.

Figures 8A and 8B show the irregular marginal contour and boggy papilla with a large fibrous mushroom clot covering the bicuspids and extending around all of the posterior teeth on the right side of the mandible. Cotton packs were found in the gingival sulcus in the molar region. The packs and clots were removed and the area was cleaned with sponges and hydrogen peroxide.

Cotton ropes moistened in a saturated solution of tannic acid were packed into the gingival sulci around the teeth in the bleeding area. This stopped the hemorrhage except from the lingual of the bicuspids which continued to bleed except when direct pressure was applied over the tissue. A periodontal surgical dressing was placed over the gingiva and there was no further bleeding, Figure 8C. After 72 hours the packs were removed.

The following laboratory tests were made:

RBC 4,050,000	Hbg. 11.0 gm.
WBC 8,850	Color index 0.82
Bleeding time, 2 minutes	Coagulation time, 4 minutes 45 seconds

Since the family history was negative, the hemorrhage was not spontaneous and the gingival condition was such that hemorrhage could easily occur after instrumentation, the patient was spared the expense of additional laboratory work.

Complete periodontal treatment including gingivoplasty was performed without difficulty. The firm non-hemorrhagic gingiva obtained by treatment is illustrated in Figure 8D. The mandibular incisors are ligated with stainless steel wire and acrylic for temporary stabilization.

SUMMARY AND CONCLUSION

Periodontal management of the abnormal bleeder has been discussed and clinical cases reported. Hemorrhagic lesions of systemic and local origin have been illustrated. Bleeding abnormalities causing trouble to patient and dentist—here reviewed—include hemophilia, purpura, polycythemia, and related states. These diseases affect periodontal structures and their management requires close physician-dentist cooperation.

4121 Camp Bowie Boulevard

BIBLIOGRAPHY

1. Wentz, F. M., Anday, D. J., and Orban, B.: Histopathologic Changes in the Gingivae in Leukemia. J. Periodont. 20:119 (October, 1949).

2. Wintrobe, M. M.: Harrison's Principles of Internal Medicine. Philadelphia, c. 1950, Blakiston, p. 267.

3. Quick, Armand J.: The Pathological Physiology of Hemorrhagic Conditions. Bulletin of the New York Academy of Medicine, abs. in D.D.S., 2:320 (May, 1953).

4. Aggeler, Paul M., and Lucia, S. P.: Hemorrhagic Disorders; A Guide to Diagnosis and Treatment. Chicago, c. 1949, University of Chicago Press, Preface, p. vii.

5. Andujar, John J.: Personal Communications.

6. Hill, Joseph M., and Haberman, Sol.: Personal Communications.

7. Merskey, C.: Hemophilia Occurring in the Human Female. Proceeding of Third International Congress of the International Society of Hematology, New York, C. 1951, Grune and Stratton, pp. 441-447.

8. Fowler, Willis M.: Hematology. New York, c. 1949; Paul B. Hoeber, 2nd edition, p. 258.

9. Kerr, Donald A.: Southwest Society of Dental Medicine, April, 1953.

10. Goldman, Henry M.: Periodontia. St. Louis, c. 1953, C. V. Mosby, 3rd edition, p. 104.

11. Vastine, A. B.: Research in Subgingival Therapy. J. Am. Dent. A., 17:507-511 (March, 1930).

12. Orban, Balint: Periodontal Disturbances; Practical Management. J. Am. Dent. A., 35:545-556, 1947.

13. Kolmer, J. A.: Clinical Diagnosis by Laboratory Examination. New York, c. 1944, D. Appleton-Century, p. 678.

14. Sodeman, Wm. A.: Pathologic Physiology; Mechanism of Disease. Philadelphia, c. 1950, W. B. Saunders, p. 451.

15. Stones, H. H.: Oral and Dental Disease. Baltimore, c. 1951, Williams and Wilkins, 2nd edition, pp. 917-920.

16. Ogilvie, R. F.: Pathological Histology. Baltimore, c. 1951, Williams and Wilkins, 4th edition, p. 101.

17. Thoma, Kurt H.: Oral Pathology. St. Louis, c. 1950, C. V. Mosby, 3rd edition, p. 1477.

18. Burford, W. N., Ackerman, L. V., and Robinson, H. B. G.: Multiple Hemangiomas of the Oral Cavity. Am. J. of Orth. and Oral Surgery, 30:395 (July, 1944).

19. Lever, Walter F.: Histopathology of the Skin. Philadelphia, c. 1949, J. B. Lippincott, p. 41.

20. Wintrobe, M. M.: Clinical Hematology. Philadelphia, c. 1951, Lea and Febiger, 3rd edition, p. 255.

21. Spies, Tom D.: Rehabilitation Through Better Nutrition. Philadelphia, c. 1947, W. B. Saunders, pp. 36-39.

22. Tisdall, Frederick F., and Jolliffe, N.: Clinical Nutrition. New York, c. 1950, Paul B. Hoeber, p. 596.

23. Crandon, T. H., et al.: Experimental Human Scurvy. New England, J. Med., 223:353, 1940.

24. Burket, L. W.: Oral Medicine; Diagnosis and Treatment. Philadelphia, c. 1952, J. B. Lippincott, 2nd edition, p. 347.

25. Schluger, Saul: The Etiology and Treatment of Vincent's Infection. J. Am. Dent. A., 30:524-532 (April, 1943).

26. Schluger, Saul: Necrotizing Ulcerative Gingivitis in the Army; Incidence, Communicability and Treatment. J. Am. Dent. A., 38:174-183 (February, 1949).

27. Bennett, J. B., and Prichard, John F.: Stomatitis with Aplastic Anemia Occurring During the Treatment of Syphilis with Neoarsphenamine. Texas State Journal of Medicine, 29:40 (May, 1933).

28. Beube, Frank E.: Periodontology. New York, c. 1953, Macmillan, pp. 3-9.

Periodontal Osseous Surgery: Osteoplasty and Osteoectomy

by NATHAN FRIEDMAN, D.D.S.,* *Beverly Hills, California*

THE principle of osseous resection was conceived and presented by Schluger[1,2] to overcome a serious shortcoming in the existing periodontal surgical techniques in certain aspects of case management. This principle grew out of repeated failures to maintain the results achieved by gingivectomy. Some types of pockets (which will be described later) treated by soft tissue excision recurred almost to their original depth. This is due to the fact that bone, while viable and healthy, may assume bizarre shapes such as notches, ledges, and jagged peaks because of periodontal disease. The gingiva does not behave in this manner (except in occasional gingival clefts), nor will it maintain such shapes if they are created surgically. Generally, even though deep pockets exist, the gingiva follows a scalloped pattern of curves. The underlying bone, however, may have an extremely irregular contour vastly different, and even opposite from the gingiva covering it (Fig. 1). If a gingivectomy were to be performed following the pattern of the bone as outlined in Figure 1, the irregularities created in the gingiva would not be maintained. It is probable that in time the soft tissue would round out and tend to revert to its original scalloped pattern and pocket depth because of soft tissue proliferation. To be successful in such cases graceful curves and slopes must be created instead of notches and irregular gingival outline. To achieve this result osseous surgery is required.

Further need for osseous surgery became apparent when Goldman[3] conceived the principle of gingivoplasty to establish physiologic contour of the gingiva. Once again anatomic discrepancies between the gingiva and the underlying bone revealed that in many instances it was necessary to recontour the bone in order to create and maintain the desired gingival architecture.

*Associate Professor of Periodontology and Oral Pathology, School of Dentistry, University of Southern California.

This paper is to be presented at the 1955 meeting of the Academy of Periodontology.

POCKET ERADICATION

It is not the purpose of this paper to justify or rationalize the concept of pocket elimination to achieve a successful result in therapy. However, this paper is predicated on the belief that a higher percentage of successful results will be obtained if pockets are eliminated in periodontal treatment. When this is possible by non-surgical means it should be done, but when a crevicular depth beyond 2-3 mm. remains following such treatment, surgical techniques should be utilized to gain the desired result.

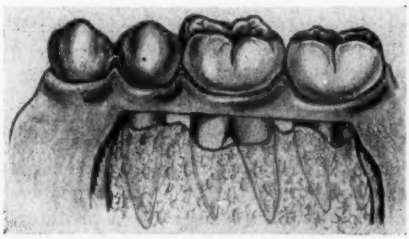


Fig. 1. The bony profile may be markedly different from that of the gingiva which maintains its normal scalloped appearance in spite of pocket formation due to periodontal disease.

Pocket elimination may be accomplished by various means, and in severely involved periodontal cases it may be necessary to employ all of them to achieve maximum results. These techniques are:

1. *Subgingival curettage*—as a result of which the gingivae recede to an acceptable crevicular depth because of the elimination of inflammation and edema.

2. *Reattachment*—the organic connective tissue union of the periodontium and the tooth is achieved with new bone,

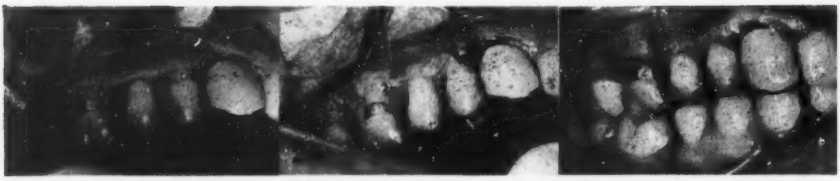


Fig. 2. 2a—Flap back—Note blunted bony crests between the bicuspids and molars and thick ledge on buccal of the first molar (arrow). 2b—Blunted bony crests reshaped to conical crests, and thick buccal ledge to thin edge. Interradicular grooving created. 2c—One week later—Note how the overlying gingiva reflects the bony changes. The interproximal papillae are cone-shaped and the gingiva reflects the interradicular grooving in the bone. In the unoperated area between the cuspid and bicuspid the contour is unphysiologic.

cementum, and periodontal membrane formation if the attempt is successful.

3. *Gingivectomy*—surgical removal of the unsupported gingiva to the base of the pocket.

4. *Osseous resection*—surgical removal of the gingiva and reshaping of the bone to eliminate the pocket and correct unphysiologic architecture.

Each of these techniques is indicated in specific situations. Where the diagnosis reveals that sufficient recession to eradicate the pocket will result from subgingival curettage, this is the indicated procedure. If reattachment attempts are deemed advisable because of favorable pocket morphology, such attempts are made and if reattachment fails, osseous surgery or compromise is indicated. When pockets are very deep or the gingiva is fibrous and thick, gingivectomy is the treatment of choice, but there are many situations where this surgical procedure is inadequate and osseous surgery must be employed. Correct diagnosis of pocket morphology by correlated clinical and roentgenographic examinations in each case will lead to the selection of the technique best suited to eliminate the pocket and, at the same time, create physiologic contour of the bone and its overlying gingiva.

TERMINOLOGY

The terms osseous resection and osteoplasty have been used interchangeably[2,4] and are apt to be confusing, especially in the light of such distinguishing terms for soft tissue surgery as gingivoplasty and gingivectomy. It was thought advisable by the author, for the purpose of clarification, to employ equivalent terminology for osseous surgery. Gingivoplasty has been defined as a plastic surgical procedure to contour or reposition the gingiva to achieve a physiologic gingival contour.[3] Gingivectomy is an operative procedure consisting of the removal of the soft tissue side of the pocket, indicated in the gingival or periodontal pocket where there is no bone in the lateral wall. Analogously, in this paper the term osteoplasty is used to describe a plastic procedure in which the periodontal pocket is eliminated and the bone reshaped in order to achieve physiologic contour of the bone and the gingiva overlying it. In this operation the bone that is reshaped is not part of the attachment apparatus, thus no bony support of the tooth or teeth is lost. Osteoectomy is an operative procedure in which bone which is part of the attachment apparatus is removed in order to eliminate a periodontal pocket and establish gingival contour that will be maintained. Osteoectomy, then, will require the loss of some bony support of the tooth or teeth, and the amount involved will be an important criterion for its use.

OSTEOPLASTY

Indications:

I. *Deep interproximal pockets on posterior teeth involving the buccal interdental bone:* These are the most common sites where osteoplasty is indicated. Figure

2A illustrates such a case. A gingivectomy had been performed previously, but the pockets were not completely eliminated nor physiologic contour achieved because of the blunted interdental bone. Note that between the bicuspids, and the bicuspid and molar, the crests which normally would be somewhat cone-shaped now resemble truncated cones. In performing a gingivectomy here the overlying gingival papillae would have to be flat to eliminate the pockets completely. Between the molars, in addition to the blunted bony crest, there is an irregular wavy outline. This is a common deformity in bone as a result of periodontal disease. Figure 2B shows the bone recontoured to create a physiologic cone-shaped alveolar crest so that the overlying gingiva will assume the same contour. In addition to the reshaping of the crests, interradicular grooving was accentuated to create root prominences so that the overlying attached gingiva would have the vertical grooves which act as spillways for food during mastication. This grooving is important for another reason. The accentuated root eminences maintain the surgically created gingival recession more successfully than when no such growth is performed. Figure 2C shows the gingiva one week postoperatively when the pack was changed. Note that the gingiva has taken the form of the underlying bone. The papillae are cone shaped and placed interproximally so that they are protected by the contours of the teeth. The vertical grooves created in the bone are reflected in the overlying attached gingiva and the entire picture is one of a sculptured normal physiologic periodontium. The area between the cuspid and bicuspid which was not operated, offers a contrast of bulk and thickness compared to the areas posterior to it.

II. *Pockets on the buccal, lingual, and palatal surfaces where resorption of the bone results in thick ledges:* This occurs quite frequently in the posterior region although occasionally anterior teeth are involved, particularly where they are in lingual version. In Figure 3A note that on the normal side the alveolar crest is thin and pointed but the bone becomes thicker apically. On the side depicting the pocket, the gingival margin is in a relatively normal position and of normal shape but the underlying bone has resorbed, creating a thick ledge. In order to eliminate this pocket and acquire physiologic contour it is necessary to do an osteoplasty. This condition may co-exist with that described in the above section and the two deformities corrected simultaneously as illustrated in the molar region in Figure 2.

Often the bifurcations of lower molars and the buccal aspect of the trifurcations of the upper molars are involved similarly and they are treated as if they were interproximal areas. The osteoplasty is performed so that the thick ledge at the bifurcation is thinned and a pointed papilla-like shape is given to the bone between the roots. The

Fig. 3. 3a—Pocket on the buccal—bone resorption resulted in a thick ledge. To eliminate the pocket and create physiologic contour an osteoplasty must be performed as indicated by the dotted line. Note on normal side the thin edge of the gingiva and the corresponding shape of the underlying bone. 3b—Failure of a gingivectomy—eight months postoperative. Note the breakdown over the cuspid which is in lingual version. The thick bony ledge on the labial should have been recontoured by osteoplasty and interradicular grooving created. 3c—Osteoplasty performed. Recontouring of the marginal and interproximal gingiva and the underlying bone was done solely with diamond stones.

interradicular septum is "tucked" between the roots by hollowing out the bone slightly between the roots, thus permitting the overlying gingiva to fall between them like an interproximal papilla. The free gingiva is thus positioned so that it is protected by the bulging contour of the crown. During mastication, food will not strike the papilla but will glance off the attached gingiva which is designed to accept and be stimulated by this frictional action.

Figure 3B illustrates a case where a gingivectomy had been performed to eliminate the pockets around the central, lateral, cuspid and bicuspid. Due to the lingual version of the cuspid a thick ledge of bone existed on the labial, and although the pocket was eliminated, this treatment proved to be inadequate. The resultant contour was not physiologic, and as a consequence the thick ledge was subjected to the trauma of food impingement. This trauma, together with the tendency of gingiva to proliferate when left in thick ledges, eventually led to the breakdown over the cuspid. This failure necessitated reoperating and an osteoplasty was performed by the use of diamond stones. The result is shown in Figure 3C.

III. *Tilted Lower Second Molar:* Frequently pockets exist on the mesial surface of the second molar where the first molar had been extracted and not replaced. Usually the second molar migrates and tilts anteriorly to various degrees. The pockets which develop are of the infrabony type, since the saddle area medial to the tooth does not resorb but the bone adjacent to the tooth does. In the illustrations shown the pocket depths ranged from five to nine mm. If one were to attempt the eradication of these pockets by gingivectomy, a deep V-shaped gash would be made. Postoperatively the wound would proliferate from the bottom of the notch and tend to reestablish the previous level and contour. In order to overcome this tendency and prevent failure it is necessary to shape the underlying bone into as long a ramp as possible, so that the overlying gingiva will take a long graceful slope as it approaches the tooth. This reconstructed architecture, while not natural, maintains itself very well and prevents the failure inherent in soft tissue surgery in these cases.

While some of these infrabony pockets may lend themselves to attempts at reattachment, others do not. This conclusion was reached after careful scrutiny of many cases. It was noted that as the tooth tilted, the relationship of the cemento-enamel junction to the bone changed until the junction was apical to the alveolar crest. It became obvious that reattachment attempts were not feasible under such circumstances since connective tissue reattachment to enamel is not possible. Therefore, each case had to be evaluated to determine which mode of therapy was indicated. Figure 4A illustrates a case where reattachment was attempted but failed. The pocket originally was 8 mm. in depth. A Hirschfeld diagnostic silver point[5] was inserted to indicate (1) the level of the gingiva in the roentgenogram, (2) which portion of the pocket is in soft tissue and which in bone, and (3) the relationship of the base of the pocket to the alveolar crest. The point is 7 mm. long and indicates that 3 mm. of the pocket is in soft tissue and 4 mm. in bone. Osteoplasty was performed and the result is shown in Figure 4B. Figure 4C illustrates a case where osteoplasty is required because the cemento-enamel junction is apical to the alveolar crest of the adjacent saddle area. The pocket depth here was 5 mm. and observation revealed that the gingiva on the mesial maintained its height so that as the tooth tilted the mesial surface was covered by the gingiva up to the marginal ridge. The subgingival calculus is on the enamel and the tissue relationships are altered entirely. Figure 4D shows the postoperative result eighteen days later. A similar but more severely involved case is illustrated by Figure 4E where the pocket depth was 6 mm. The cementoenamel junction was below the alveolar crest and although it might have been feasible to attempt partial reattachment, osteoplasty was considered to be the treatment of choice (Fig. 4F).

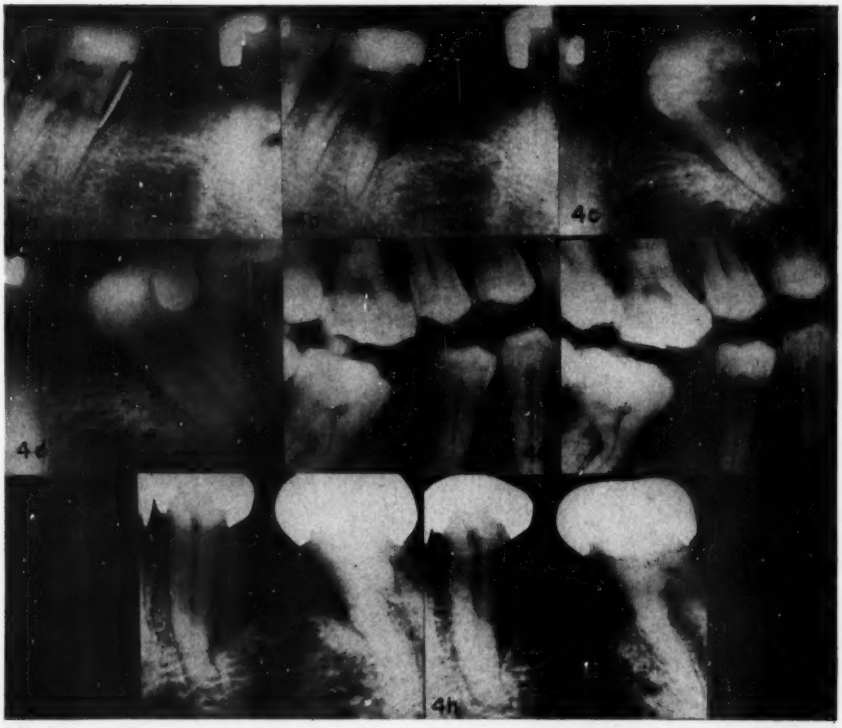


Fig. 4. 4a—Tilted lower molar—Infrabony pocket where reattachment had been attempted and failed. Hirschfeld point indicates a 7 mm. pocket still exists. 4b—Osteoplasty immediately postoperative to show the ramping of the bone to permit a long gradual slope of the overlying gingiva. 4c—Reattachment should not be attempted because the cemento-enamel junction is apical to the bony crest. Pocket must be eliminated by osteoplasty. 4D—Postoperatively eighteen days—Note the long ramp of the bone. 4e—Osteoplasty indicated because cemento-enamel junction is apical to bony crest, although partial reattachment is possible. 4f—Pocket eliminated and bone ramped by osteoplasty. 4g—9 mm. pocket on distal of bicuspid extending around to buccal and lingual. 4h—Osteoplasty performed—eight months postoperative.

At times, one may be in doubt as to the advisability of saving a tooth. Several factors will influence this decision as well as the prognosis of the tooth involved and the selection of technique to be used in the eradication of the pocket. These factors are (1) the amount of bone loss, (2) pattern of bone loss, (3) mobility of the tooth, (4) importance of the tooth for restorative dentistry, (5) ability to eradicate the pocket. Figures 4G and 5 are cases in point. Correlated roentgenographic and clinical examinations revealed that in Figure 4G there was a 9 mm. pocket on the distal of the lower left second bicuspid which extended around to the buccal and the lingual and morphologically was not conducive to a reattachment attempt. On the mesial and mesio-buccal the bone was at a normal level. The molar was involved similarly but to a lesser degree. Although the bicuspid was considered to be a borderline case, consideration of the above mentioned factors led to the decision to attempt treatment. About half of the attachment was destroyed by periodontal disease but the pattern of the loss was considered favorable because with normal alveolar crest height on the mesial, more attachment remains than if the pattern of bone loss had been circumferential. The mobility was moderate, the pocket was eradicable by

Fig. 5. 5a—Preoperative roentgenogram. 5b—Postoperative roentgenogram (osteoplasty) note no loss of supporting bone. 5c—Preoperative—9 mm. pocket on the mesial of the right cuspid. 5d—Flap back exposing a bony well. Decision to do an osteoplasty rather than attempt reattachment was made at this point. 5e—Thin wall of the well eliminated and bone ramped labially and mesially. No bony support has been removed. 5f—Postoperative result of the osteoplasty.

osteoplasty, and this tooth as well as the molar was considered to be important to the restorative plan. Although the prognosis was guarded, evaluation after the treatment led to a revision of the prognosis to favorable. The loss of these two teeth would have necessitated the construction of a unilateral partial since the first molar

was the only missing tooth in the lower arch. Such an appliance is an awkward one at best, and is to be avoided if possible.

The case illustrated by Figure 5 is included in the tilted molar category because of the similarity of the problem and its solution. A 9 mm. pocket was present on the mesial of the cuspid (Fig. 5C). Again, the five factors listed above were considered in reaching the decision to treat, the technique to be used, and the projection of the prognosis. The amount of the bone loss was considerable but the pattern was favorable. Noting that the alveolar crest height was normal on the distal of the cuspid, arcing down on the lingual toward the mesial, it was thought that enough bony support remained to warrant the retention of the tooth. The mobility was moderate and of the same degree as the bicuspid. To these five factors was added an additional one, that of the time element. The cuspid was considered to be an important tooth for the restorative plan which called for a fixed anterior splint. The decision as to which technique to employ, osteoplasty or reattachment attempt, was postponed until the flap was laid and the actual lesion could be viewed. With the flap back (Fig. 5D) the decision was made to perform an osteoplasty because of the morphology of the lesion, coupled with the realization that if reattachment were attempted several months would have to elapse before the restorative plan could be implemented, and should the attempt fail it would have to be repeated or an osteoplasty done at that time. Hence, the thin wall of the bony well was removed and the bone ramped to a gradual slope in a labial direction and toward the mesial as well (Fig. 5E). The postoperative roentgenogram is seen in Figure 5B and the clinical result in Figure 5F. In both of the cases, as represented by Figures 4G and 5, the correctness of the decisions was borne out by the postoperative results. The prescription of a fixed anterior splint was not carried out and despite the use of the cuspid for a partial denture abutment it is serving successfully after two years. There has been no recurrence of the pocket.

OSTEOECTOMY

Indications:

I. *Interproximal craters in bone:* In the development of a periodontal pocket the usual pattern of alveolar crest bone resorption is horizontal. Not infrequently, however, the bone resorbs in such a way as to result in a crater. Buccal and lingual spines of bone remain while the interproximal portion is lost. The gingiva over it, although detached interproximally, is cone shaped and at its normal height. However, clinical examination by probing reveals that there is no detachment at the buccal and lingual line angles so that the pocket is confined to the interproximal area. Sometimes the roentgenographic examination correlated with the clinical will lead to a diagnosis of an interproximal bony crater, but very often it is difficult to determine in advance the exact morphology of this type of pocket.

There are two ways to eradicate such a pocket: by reattachment, or osteoectomy. Figure 6A illustrates a bony crater with a periodontal probe in position to indicate that the bottom of the pocket is within the crater and apical to the buccal and lingual spines of the alveolar crest. A reattachment attempt is feasible in this type of pocket since the protection for the blood clot is provided by four hard walls, the bone on the buccal and lingual, and the teeth on the mesial and distal. However, success demands a large amount of new tissue to be formed with reattachment of periodontal fibers to both teeth. Such an attempt should be made in the anterior part of the mouth, especially where esthetics is important. Figures 6D and 6E are preoperative and postoperative roentgenograms of a successful attempt at reattachment where a crater existed between anterior teeth. Reattachment should be attempted also in those posterior areas where success seems probable. The deeper the crater, the more reluctant one would be to perform an osteoectomy and inclined to attempt reattachment. Conversely, the shallow but wide crater would be less favorable for reattachment and an indication for an

Fig. 6. 6a—Crater in bone where the overlying gingival papilla is normal in shape and position. 6b—Osteoectomy performed to eliminate the crater by removing one spine of bone. 6c—Postoperative diagram to show ramp of the bone and the overlying gingiva. 6d—Preoperative roentgenogram of bony crater. 6e—Reattachment attempt successful in eliminating the crater. Note (arrow) the ledge created by curettage of the root. 6f—7 mm. pocket exists between the bicuspid and molar with gingival contour and position relatively normal and 6 mm. pocket between molars with crater of the interproximal papilla. 6g—Flap back reveals crater in bone between the bicuspid and molar and blunted bony crest between the molars. Supporting bone had to be removed over the mesio-buccal root of the molar and the distobuccal of the bicuspid (osteoectomy). 6h—Postoperative. Note ramping and long graceful curve created by osteoectomy and the results of the osteoplasty between the molars.

osteoectomy. However, it must be recognized that if reattachment is attempted and fails, osteoectomy is the only technique with which the pocket can be eliminated.

When doing an osteoectomy the crater is eliminated by removing one of the spines and ramping the bone either to the buccal or the lingual. The choice of which spine to remove will depend on several factors: such as access to the operative field, esthetics, the morphology of the crater, and which surface will be more accessible to the patient for physiotherapy. It is important that this last factor be kept in mind since the architecture resulting from the osteoectomy, although physiologic, is not normal. Figure 6B shows one wall of the crater removed and Figure 6C shows how the overlying gingiva will follow the bony contour, creating a ramp but eliminating the pocket. Figure 6F is a preoperative picture where a 7 mm. pocket existed between the upper left second bicuspid and first molar, and a 6 mm. pocket between the upper left first and second molars. Note that the gingival papilla between the upper left second bicuspid and first molar is cone shaped and almost in normal position, while the papilla between the upper left first and second molars is blunted and cratered. Yet with the flap back (Fig. 6G) we see that there is a bony crater between the bicuspid and molar, and a blunted bony ledge between the molars. The former requires an osteoectomy, the latter an osteoplasty. These were performed, the bone being ramped to the buccal in the osteoectomy, and good interproximal contour was achieved by osteoplasty (Fig. 6H).

II. *Extremely deep interproximal pockets:* This is not a common situation but

Fig. 7. 7a—Postoperative result of a gingivectomy performed where a very deep interproximal pocket existed. Tissue has proliferated and pocket recurred. (Courtesy of Dr. Saul Schluger) 7b—Preoperative roentgenogram of a 10 mm. pocket. 7c—Clinical appearance following subgingival curettage. 7d—Flap back showing a slight crater in bone filled with granulation tissue. 7e—Osteoectomy performed eliminating crater and creating interradicular grooving. 7f—Postoperative. Long ramp created from the lingual to the buccal and gradual curve of gingiva resulted instead of a V-shaped notch.

when the operator is confronted with it the only solution for successful pocket eradication lies in osteoectomy. It was the repeated failure with gingivectomy in such cases that led Schluger to think in terms of osseous surgery. Figure 7A is a postoperative result by gingivectomy. The deep V-shaped gash filled in from the base and resulted in the recurrence of the pocket to a depth of 7 mm. The original pocket was 11 mm. deep.

Figure 7B is a roentgenogram of a similar case. This pocket was 10 mm. in depth. It presented a problem because the bone on the distal of the bicuspid was intact as was the crest on the mesial of the cuspid. The patient had a full complement of natural teeth and the loss of these two teeth would break the continuity of the arch and entail a complicated restoration. Esthetics was not a problem since the cheek was heavy, the lip long, and in speech or laughing the cemento-enamel junction was not revealed. Consequently it was decided to do an osteoectomy. The area after subgingival curettage is shown in Figure 7C. The inflammation has subsided and some shrinkage of the gingiva has occurred. In Figure 7D the flap is back revealing a slight crater in bone which is filled with granulation tissue. Figure 7E shows the bone trimmed to provide a scalloped curve and interdental grooving. The crater has been eliminated and the bone ramped buccally. The postoperative result is seen in Figure 7F. Note the long ramp from the lingual and the scalloped curve of the gingiva, which will be maintained with reasonable care by the patient. The proximity of the muscle attachment to the gingival margin over the cuspid may necessitate its repositioning in the future. From the cases presented it becomes apparent that frequently a combination of bony changes occur in one operative area and both osteoplasty and osteoectomy may be performed at the same time.

III. *Shallow infrabony pockets (interproximal), and where reattachment attempts have failed*: The infrabony pocket

occurs quite frequently in periodontitis and is treated best by reattachment. Success is crowned by pocket eradication and increased bony support of the tooth, as illustrated by Figures 8A and 8B. However, if reattachment is not attempted, or the attempt is made and it fails, then the elimination of the pocket can be achieved only by osteoectomy. Figure 8C illustrates a shallow intrabony pocket on the mesial of the first molar. This was eradicated by osteoectomy (Fig. 8D). If the pocket is deep too much bone which is part of the attachment apparatus of the adjacent tooth would have to be sacrificed. Under such circumstances failure to gain reattachment would require either repeated attempts to succeed, extraction of the tooth involved, or an acceptance of a compromised result in permitting the pocket to remain. Clinical judgment will determine which course to follow.

It is interesting to note that infrabony pockets were present also on the distal of the first molar and the mesial of the second molar in Figure 8C. The eradication of these pockets would be classified as an osteoplasty, since the peak of bone removed was not part of the attachment apparatus of either tooth.

METHODS

There are three methods of performing periodontal osseous surgery. They are:

1. Modified flap
2. Full flap
3. Use of diamond stones without a flap (osteoplasty only). The armamentarium consists, in addition to that used in performing a gingivectomy, of large round burs, bone files, small bone gouge and chisel, periosteal elevator, and very coarse diamond stones of various shapes.

The modified flap is the technique most frequently used for both osteoplasty and osteoectomy. It consists of measuring the pocket depth with a periodontal probe, marking this depth on the outside of the gingiva by a puncture point, and performing a gingivectomy. Bone may or may not be encountered, depending on the case. With the gingiva removed a periosteal elevator is used to detach the periosteum and overlying gingiva from the bone, exposing it sufficiently to provide adequate access. To change the contour of the bone from a thick ledge to a thin margin, and/or the blunted interdental bony crest to a physiologic cone-shaped crest and create interradicular grooving, the instrument of choice is the #10 round bur. When performing an osteoectomy as illustrated in Figures 6G and 7D the interdental bone may be removed to the bottom of the crater by a Buck file or a bur, but the bone over the teeth must be removed with a small bone gouge or chisel. The reason for this is that the bur would be apt to cut into the roots of the teeth. Following the reshaping of the bone to the desired contour, the area is thoroughly debrided and the flap is replaced. Sometimes the flap does not completely cover the bone even though it did so prior to its elevation. This is due to the detachment of the flap and the pull of muscle fibers in the muccobuccal fold. A piece of dry foil (tin foil with an adhesive on one side) is placed over the flap, and the area covered with periodontal pack. The foil will prevent the pack from being pushed under the flap, which would complicate healing. In placing the pack it is best to do so by a light pressure in a coronal rather than apical direction, so that the flap will not be displaced. At the end of one week the foil is removed and a fresh pack is placed. Foil is not required with the second pack since the flap is firmly attached by this time, and bone that may have been exposed will be covered by granulation tissue.

The full flap technique is used infrequently since the modified flap is better suited to achieve the results in most situations. However, a full flap is laid where the decision is to be made between osseous surgery or reattachment attempt after exposing the lesion (Fig. 5D). In other cases where there are many bony changes to be made, the operator may wish to expose the

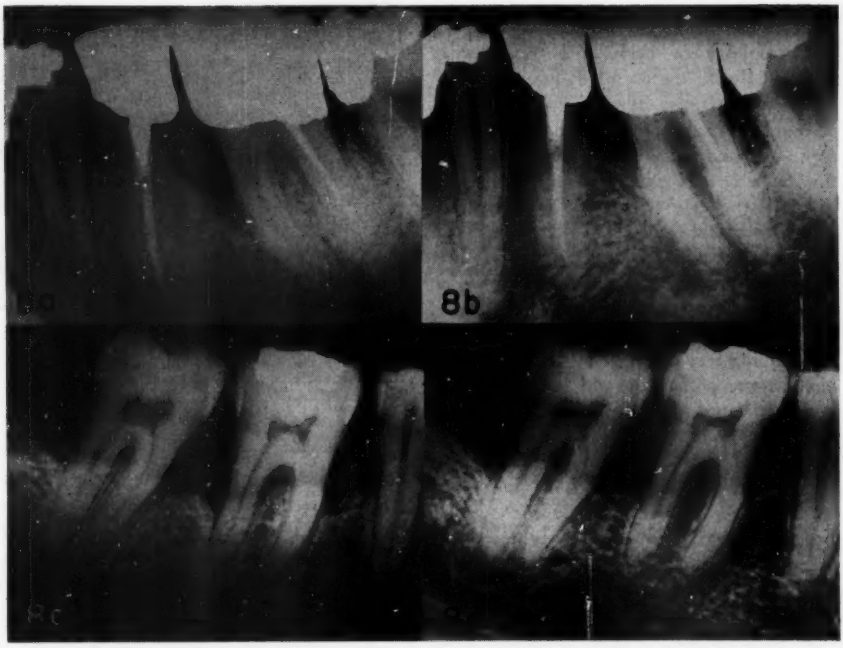


Fig. 8. 8a—Preoperative roentgenogram of a shallow infrabony pocket. 8b—Postoperative roentgenogram showing successful reattachment attempt. 8c—Preoperative roentgenogram showing a shallow infrabony pocket on the mesial of the first molar and infrabony pockets on distal of the first molar and mesial of the second molar. 8d—Postoperative result of osteoectomy between the bicuspid and molar and osteoplasty between the molars.

field to determine the extent of the osseous correction. The excision of the soft tissue in this technique is inaccurate and often results in poor gingival contour, so that it may be necessary to reoperate later by performing a gingivoplasty or gingivectomy. When laying a flap (Fig. 2A) cuts are made through the interproximal papillae mesiodistally with a small thin knife which is then used to detach the tissue on the buccal and lingual by slipping it under the gingiva down to the bone, thus severing the attachment to the teeth. Sometimes it is advisable to make a vertical incision at the anterior end of the area to facilitate lifting the flap. A periosteal elevator is used to lift the flaps on the buccal and lingual, the bony changes are made and the flaps brought back into position. Wherever excess gingiva remains it is trimmed off with a pair of fine scissors and the flap is sutured into position. No foil is necessary but it is advisable to place a pack as a protective measure.

Often in treating the tilted lower second molar a combination of modified and full flap is required. The gingivectomy may be done first, starting from the distal of the second bicuspid and creating as long and gradual a slope as possible to the mesial of the second molar. Then an incision is made over the center of the alveolar crest and the flap laid buccally and lingually. The bone is contoured to eliminate the precipitous drop on the mesial of the second molar, transforming it into a gradual ramp so that the overlying gingiva will take this form. Here the bone may be shaped by the bur and chisel, depending upon the access and tilt of the molar. As the tooth is approached the bur is not used since it will be difficult to get into the undercut and

may cut into the cementum.

Osteoplasty is performed most easily and rapidly by the use of diamond stones.[6] These stones must be very coarse and revolving at high speed to be effective. In some cases no incisions are made since the operation is performed for plastic reasons only. In this case (Fig. 3B) it was merely a matter of contour, since the pocket was eliminated previously by gingivectomy but the failure is obvious. What was started as a gingivoplasty with diamond stones ended by being an osteoplasty. As the contour of the gingiva was changed by the stone, the gingiva was perforated and the underlying bone reshaped, thus gingivoplasty and osteoplasty were accomplished without a flap. In other cases such as exemplified by Figure 3A, the pocket may be eliminated by an incision at a right angle to the tooth and the physiologic contour achieved by the diamond stone which would perforate the gingiva and contour the underlying bone. If very much bone is exposed, dry foil should be used to cover the area before the pack is placed. Although this is not as neat a surgical technique as the others, the areas heal uneventfully and excellent results are achieved.

A brief discussion of the postoperative period is in order at this point. Although more postoperative discomfort may result from periodontal osseous surgery than soft tissue surgery (by no means is this the rule), postoperative infection is rare. The explanation for this may lie in the fact that with reasonably careful surgical techniques, followed by debridement of the wound and the proper application of a pack, the protective elements of the saliva may prevent postoperative complications. Saliva possesses antibacterial agents effective against some pathogenic organisms and may have the property of converting pathogenic organisms into non-pathogenic organisms. Bibby[7] discusses additional characteristics of saliva which partially explains why the tissues of the mouth are so resistant to injury and infection. It is safe to say that if the same amount of care is taken in handling the tissues when performing periodontal osseous surgery as in other dental operations, the incidence of postoperative complications will be no higher. If the patient has exhibited poor powers of healing previously or is prone to have postoperative pain, antibiotics can be utilized prior to and/or after surgery.

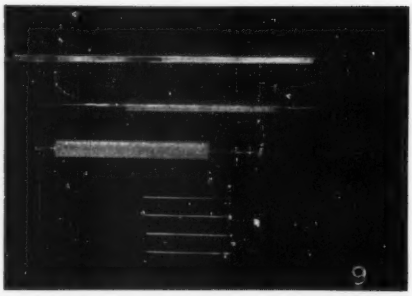


Fig. 9. Part of the armamentarium used in performing osseous surgery. From above down; Buck file, periosteal elevator, bone chisel, large round bur and the three diamond stones designed by Dr. Lewis Fox. The large wheel stone is the most effective and most often used.

SUMMARY

The need for osseous surgery in periodontal treatment grew out of repeated failures to achieve, in some aspects of case management, pocket elimination and physiologic gingival contour that could be maintained.

In soft tissue surgery the terms gingivoplasty and gingivectomy are used. In this paper analogous terminology for osseous surgery is suggested together with the definitions and the basis for using these terms. They are osteoplasty and osteoectomy, and are used to differentiate respectively between procedures where the removal of bony support of the tooth is not necessary, and those procedures which require the removal of such bone. Six indications are discussed: three for osteoplasty and three for osteoectomy. The objectives of each operation are delineated; the choice of treatment and prognosis are discussed.

Attempts to eliminate infrabony pockets by reattachment sometimes succeed (the degree of success seems to vary with dif-

ferent operators). Where there is failure, such pockets can be eliminated only by osseous surgery. The correct diagnosis of pocket morphology (especially in the tilted lower second molar) will lead to the selection of the correct technique. The illustrations are used to point this out and show the results obtained by osseous surgery.

Two methods of performing osteoplasty and osteoectomy are described and the armamentarium is illustrated. A third method of performing osteoplasty by the use of diamond stones is presented.

Postoperative complications are rare and the probable reasons are given for the resistance to infection.

This concept represents a basic principle in periodontal surgery. The application of this concept is necessary to overcome the shortcomings inherent in soft tissue surgery under certain circumstances.

405 North Bedford Drive.

(I wish to acknowledge my appreciation to Carol E. Cooke, D.H. for the drawings.)

REFERENCES

1. Schluger, Saul: Osseous Resection—A Basic Principle in Periodontal Surgery. Oral Surgery, Oral Medicine, and Oral Pathology, Vol. 2:316, 1949.

2. Schluger, Saul: Surgical Techniques in Pocket Elimination. Texas D.J. 70:246, 1952.

3. Goldman, H. M.: The Development of Physiologic Gingival Contours by Gingivoplasty. Oral Surgery, Oral Medicine, and Oral Pathology, 3:879, 1950.

4. Goldman, H. M.: Periodontia ed. 3, St. Louis, C. V. Mosby Co., 1953.

5. Hirschfeld, L.: A Calibrated Silver Point for Periodontal Diagnosis and Recording. Journal of Periodontology, 24:94, 1953.

6. Fox, Lewis: Personal Communication.

7. Bibby, B. G.: What About Saliva? Oral Surgery, Oral Medicine and Oral Pathology, 2:72, 1949.

DR. DANIEL PRESTON MOWRY, 1894-1955

Members of the American Academy of Periodontology were saddened to learn of the death of Dr. Daniel Preston Mowry on August 2, 1955. Dr. Mowry was dean of the faculty of dentistry of McGill University and professor of periodontia. He was a past president of the Montreal Dental Club, a Fellow of the American College of Dentists and the Royal College of Surgeons, as well as a member of the American Academy of Periodontology. He was elected twice as president of the College of Dental Surgeons of the province of Quebec.

Although very active in many dental organizations, he found time to devote to civic affairs, serving a term on the City Council of Montreal and the City Board of Health of Montreal. He was also a member of the Board of Governors of Sir George Williams College and a past president of the St. James Literary Society.

Dr. Mowry was noted for his spirited comments on the importance of periodontology which he made frequently at dental meetings. He is survived by his widow, one son, Stephen, who is now in California, and two grandchildren.

Experimental Occlusal Trauma

by S. N. Bhaskar, D.D.S., PH.D., and Balint Orban, M.D., D.D.S., *Chicago, Illinois*

THE first description of excessive occlusal stresses and their relation to dental disease was given more than fifty years ago.[1] It was then claimed that undue stresses on the teeth may lead to "pyorrhea." Since that time, numerous reports concerning the subject have appeared in the literature and these fall into two main groups. According to one group, excessive occlusal stresses, also called occlusal trauma,[2] traumatic occlusion, traumatogenic occlusion,[3] or trauma from occlusion,[4] do not cause inflammatory periodontal diseases such as gingivitis and periodontitis,[4-18] but produce a series of specific changes in the supporting structures of the tooth. These changes vary on the sides of pressure and tension. On the pressure side, resorption of bone, necrosis of periodontal fibers, hemorrhages, thrombosis of blood vessels, and in some instances, resorption of cementum occurs. On the side of tension, the periodontal membrane fibers are stretched, and bone is laid down on the socket wall.[4, 5, 7, 8, 9, 11, 16, 17] These reports claim that excessive occlusals stresses, the majority of which are really lateral forces, do not cause downgrowth of the epithelial attachment. The evidence for these views is based on a study of the effects of placing high crowns on the teeth of dogs,[11, 16] monkeys and rats,[11] and analyses of serial histologic sections of human jaws[5, 9] in which teeth had been extracted but not replaced.

The second group of reports claim that occlusal trauma or excessive occlusal stresses produce one or a number of conditions which include: Gingivitis, alteration of gingival contour and production of McCall's festoons and Stillman's clefts, formation of pockets, calculus deposits, erosion, caries, reticular atrophy of pulp, pulp stones, some cases of "tic douloureux," temporomandibular joint disturbances and the formation of disastemas, etc.[19-32] Experimental evidence for the claim that excessive occlusal stresses cause periodontal diseases is based mainly upon two investigations. Box,[25] after cementing a crown on a lower incisor of one sheep demonstrated the formation of excessive calculus on this tooth and showed that the lingual or the tension side of the tooth developed a pocket. The second experimental evidence which might favor this latter view is that of Stones',[27] who placed cement and silver amalgam fillings in three adjoining posterior teeth of seven monkeys. One of these fillings was left higher than the other two and the animals were sacrificed from 10 to 43 weeks after the restorations had been placed. Histologic sections, according to Stones, showed signs of definite periodontal disease in three monkeys; some changes in another three; one monkey showed no change. The "histologic signs of definite periodontal disease" was the growth of epithelial attachment along cementum surface and the presence of inflammatory cells in the gingival connective tissue. In the control animals, the author claimed, however, that the epithelial attachment "appeared as a smooth band which joined neatly onto the cementum." Although no pockets were reported, the author assumed that vertical type of pockets might have developed.

There are several experimental papers published which offer proof that traumatic occlusion does not lead to gingival disturbances.[33, 34, 35] In view of these conflicting opinions in the literature, the present investigation was undertaken to determine once more whether excessive occlusal stresses had any relationship to periodontal disease or to inflammation in the gingiva and periodontal membrane.

MATERIALS AND METHODS

Four Rhesus macacus monkeys, 4½ to 6 years of age, with permanent dentitions, were anesthetized, given thorough prophylaxes. MOD cavities were prepared in the

upper right first premolars. In preparation of the cavities, the buccal cusps were beveled. Compound impressions were then taken in copper bands and stone dies were made. On the dies, wax patterns were made in such a manner that the buccal cusp and its inclined plane was exaggerated. The wax impressions were then inserted in the actual cavity, adapted, tested in occlusion to see that the buccal cusps were "high." The cast inlay was cemented to place, polished and adjusted so that in the centric and the right lateral movements, the tooth was subjected to excessive occlusal stress.

The two younger monkeys (4½ yrs.) were sacrificed at three days and three weeks, while the two older ones (6 yrs.) were taken at three months and six months. All animals were profused with 10% formalin; thier jaws decalcified and the upper right premolar areas was sectioned serially in a buccolingual direction while the corresponding region of the lower jaw was sectioned in a mesiodistal direction. The left side, which was used as the control, was sectioned similarly. The sections were stained with hematoxylin and eosin, some with the Mallory method; others by silver impregnation.

Fig. 1A. Three-day experiment. Photomicrograph of a bucculingual section through first premolar. A portion of the curved canine can be seen palatal to the buccal root of the premolar.

At the beginning of the experiment and at various intervals during the experimental period, Kodachrome pictures were taken of both the control and experimental sides. This was done to record objectively any changes that might have occurred in the gingiva. Roentgenograms of the experimental side were taken before and at the termination of the experiment.

THREE DAYS (MONKEY #4)

Findings: Three days after cementing the inlay, the tooth moved slightly buccally so that on the control side, the opposing teeth which previously were about lmm. apart, now came almost into contact. Histologic sections of the experimental tooth showed the following changes:

Buccal side: The apical end of the epithelial attachment was at the cemento-enamel junction and the bottom of the sulcus was on the enamel. The free and the attached gingiva showed isolated lymphocytes. In the coronal third of the buccal plate, the tooth surface was in close proximity to the bone; the intervening periodontal membrane was compressed and hyalinized and the periodontal blood vessels showed thrombosis (Figs. 1 & 2). The alveolar bone in this pressure area showed extensive resorption lacunae and osteoclasts. There was a widening of the periodontal space in the region of the apical two-thirds of buccal plate (Figs. 1 & 3) and although the collagenous bundles of the periodontal membrane seemed intact, numerous periodontal blood vessels showed thrombosis (Fig. 3). The periodontal surface of the alveolar bone showed numerous areas of resorption by osteoclasts.

Palatal side: The apical end of the

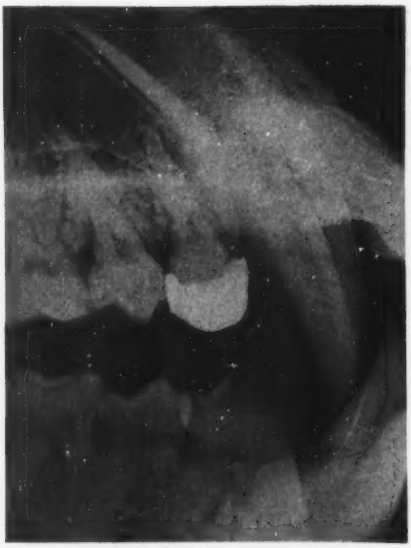


Fig. 1B. Roentgenogram showing the relationship of the cuspid root to the bicuspid roots.

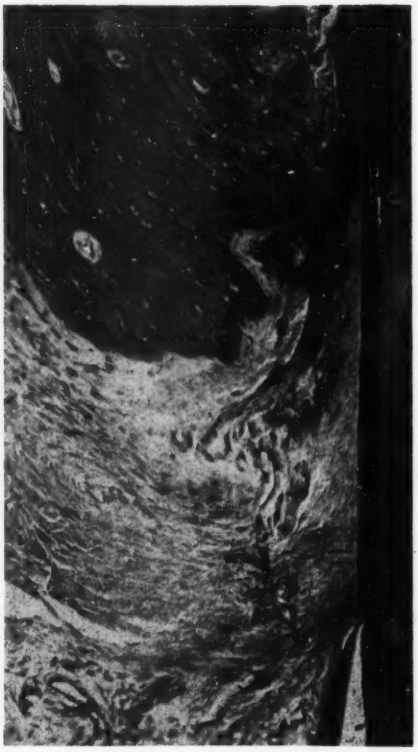


Fig. 2. Three-day experiment High power of area A in Figure 1A (pressure side) a. Necrotic periodontal membrane; b. Cementoenamel junction.

epithelial attachment was at the cemento-enamel junction and the bottom of the sulcus was on the enamel. The lamina propria of the free and the attached gingiva showed few isolated lymphocytes. The periodontal membrane in the region of the coronal half was widened; the principal fibers appeared stretched; and the bone surface showed a thin seam of osteoid (Figs. 4 & 5). In the apical half, the fibers were relaxed and the periodontal surface of the bony plate did not show any signs of resorption.

Inter-radicular area: The tooth impinges the periodontal membrane upon the crest of the inter-radicular bony septum (Figs. 1A & 4). The intervening connective tissue was partly destroyed and hyalinized. The blood vessels in this area showed thrombosis (Fig. 6). The coronal half of the palatal surface of the buccal root, and the apical half of the buccal side of the palatal root were tension areas. In these regions, however, the periodontal membrane, though widened, showed thrombi in the blood vessels as well as resorption of the surface of the bony socket. In areas of pressure, which were the apical part of the palatal surface of the buccal root (Fig. 3b) and coronal part of the buccal surface of the palatal root (Fig. 6a), narrowing of the periodontal membrane, crushing and necrosis of fibers, thrombosis and localized necrosis of bone could be seen.

THREE WEEKS (MONKEY #3)

Three weeks after the inlay was cemented, the first right premolar moved buccally to such an extent that the opposing teeth were almost in occlusion (Fig. 7). In other words, the experimental tooth moved almost out of trauma. Clinical examination during the experimental period and at sacrifice, did not reveal any changes

Fig. 3. Three-day experiment. Photomicrogram shows tissue changes in the area of tension (a), and pressure at apex (b). This is a high power of area B shown in Figure 1A.

Fig. 4. Three-day experiment. Photomicrogram of a buccolingual section through the palatal root of the first premolar. Tension area (a), and pressure area in bifurcation (b).

in the gingiva of the experimental tooth. The histologic sections showed the following changes:

Buccal side: The apical end of the epithelial attachment was at the cemento-enamel junction and the bottom of the sulcus was on the enamel. The free and the attached gingiva showed isolated lymphocytes. The coronal part of the periodontal membrane, which at three days was very narrow or obliterated, at three weeks appeared wider than normal (Fig. 8). The width of the periodontal membrane of the tension area in the apical half was, as at three days, greater than normal. The excessive bone destruction which was noticed on the entire alveolar wall at three days, at three weeks was markedly reduced. In dispersed areas, both in the coronal as well as the apical regions, patches of bone apposition could be seen. The periodontal membrane did not show thrombosis or necrosis. The most striking change at this stage was the presence of numerous resorption areas on the surface of the root (Figs. 8-11). These resorption lacunae involved the acellular as well as the cellular cementum and the deeper ones extended into the dentin for varying depths.

Palatal side: The apical end of the epithelial attachment was on the cemento-enamel junction and the bottom of the sulcus was on the enamel. The periodontal membrane was of more or less even width and did not show necrosis or thrombosis of blood vessels (Fig. 10). The apical half of the socket wall showed areas of resorption and in some areas close to the apex, the resorption had almost completely destroyed the original "alveolar bone proper" and in its place, osteophytic type of bone could be seen (Fig. 9). Corresponding to the areas of the bony wall where resorption could

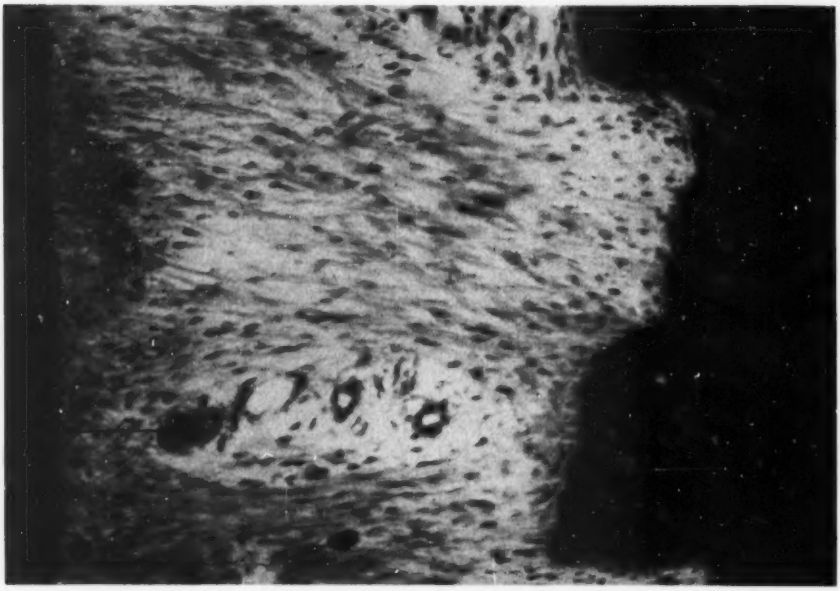


Fig. 5. Three-day experiment. High power photomicrogram of area (a) shown in Figure 4. Tension side: a, capillary hemorrhage—b, thrombosed vessel.—c, bone formation.

be seen, the root surface also showed resorption lacunae (Figs. 9 & 10). In the coronal third of the root, the tooth surface did not show resorption.

Inter-radicular area: In the area of the bifurcation itself, the tooth impinged the periodontal membrane upon the crest of the bony septum and the intervening collagen fiber bundles had undergone necrosis (Fig. 11). Some blood vessels between the tooth surface and bone and others in the marrow spaces of the bony spetum showed thrombi. In the most superficial layers of the bony septum some of the lacunae were empty and the osteocytes had apparently undergone degeneration.

On the palatal side of the buccal root, the upper half of the periodontal space was narrowed and at a few points still obliterated (Figs. 8 & 13). In the latter areas, necrosis of collagen bundles and bone could be seen. Elsewhere in this portion of the bony wall, osteoclasts were pres-

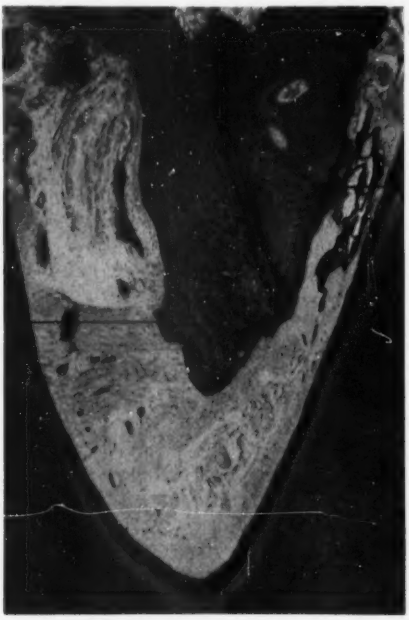


Fig. 6. Three-day experiment. Photomicrogram of the inter-radicular area. (Area C from Figure 1A—a. pressure area, b. tension area).

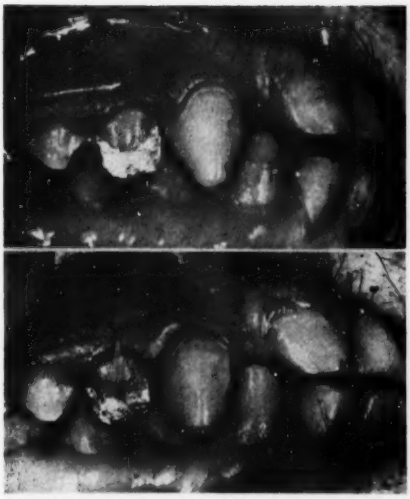


Fig. 7 Photomicrogram of the experimental side (right) of a monkey (3) soon after insertion of the inlay (A) and three weeks later (B).

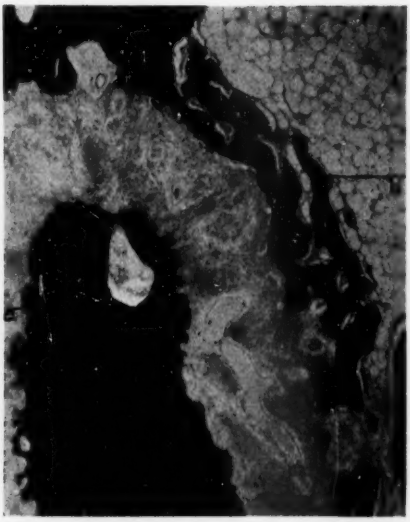


Fig. 9. Three-week experiment. High power of area shown in Figure 8. Note root resorption (a) and osteophytic bone (b) on the palatal side of the root apex.

Fig. 8. Three-week experiment. Photomicrogram of a buccolingual section through the first premolar. The buccal gingiva is missing in this section, but was present in some other sections of the series.

ent and resorption was in progress. Although a thin layer of bone apposition could be seen on the surface of the bony socket, the pattern of bone clearly indicated that in this area extensive bone destruction had occurred during the experiment. This was evidenced by the fact that the original bundle bone had been completely destroyed and in the marrow spaces, irregular osteophyte-like (immature) bone could be seen (Figs. 8 & 9). The apical half of the palatal surface of the buccal root showed areas of resorption (Fig. 9).

On the buccal side of the palatal root, the apical half showed a wider periodontal space, bone resorption on the socket wall, and absence of resorption on the surface of the root (Fig. 8). The coronal half of the buccal side of the palatal root showed narrowing of the periodontal membrane, and resorption of both the socket wall and the root surface (Fig. 12).

THREE MONTHS (MONKEY #2)

In this animal, as in the previous one,

Fig. 10. Three-week experiment. Photomicrogram of the palatal side of the first premolar—a. bottom of the gingival sulcus, b. cementoenamel junction.

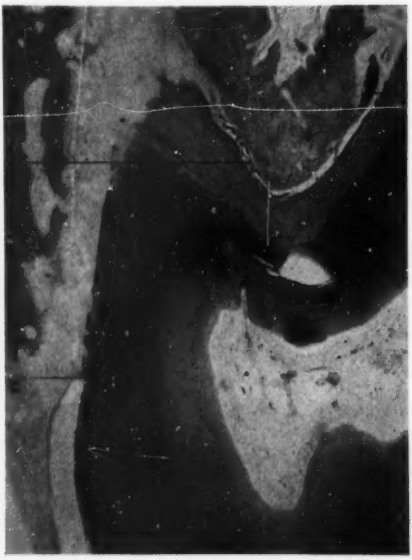


Fig. 11. Three-week experiment. Photomicrogram of the inter-radicular area (a & b)—Necrotic tissue masses in the bifurcation (c) cementoenamel junction.

the experimental tooth had moved out of the trauma and the teeth of the opposing arches were in normal functional occlusion. Clinical examination did not show any difference in the texture or coloration of the gingiva on the experimental and control sides. The microscopic changes were as follows:

Buccal side: The apical end of the epithelial attachment had migrated for a short distance onto the cementum surface but the bottom of the sulcus was still on the enamel. The same relationship was found on the control side and was apparently due to the age of the animal. The width of the periodontal membrane on this side was more or less even and normal (Fig. 14). Dense collagen bundles could be seen extending from the bony wall to the surface of the root and neither tearing of fibers, necrosis, nor thrombosis was present. The bony wall of the socket showed a consistent picture of bone apposition which was evidenced by a thin osteoid seam. That this bone apposition had gone on for some time was evidenced by the fact that the surface of the bony socket was formed by a wide layer of lighter-staining mature lamellated bone which was separated from the older bone by an irregular reversal line (Fig. 15). The latter indicated the area where the bone destruction ended. The surface of the root showed repair of the resorption areas. This repair for the most part was anatomical, i.e., the normal con-

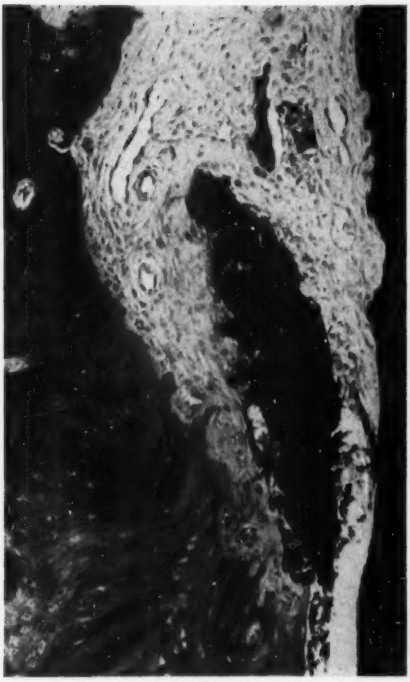


Fig. 12. Three-week experiment. High power of area (a) shown in Figure 11.

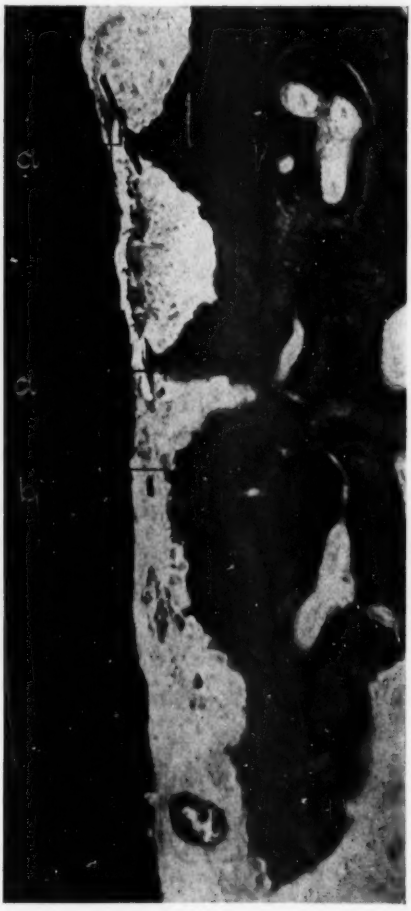


Fig. 13. Three-week experiment. High power of area (b) shown in Figure 8. Necrotic areas in periodontal membrane—(b) bone resorption in progress.

tour of the root surface was restored. The tissue of repair was cellular cementum.

Palatal side: The apical end of the epithelial attachment had migrated to the surface of the cementum but the bottom of the shallow sulcus was on the enamel. The same relationship, however, was found on the control side. The periodontal membrane, though slightly wider at the coronal end than elsewhere, was normal (Fig. 14). Dense collagen bundles could be seen extending from the bone to the tooth surface. Whereas the coronal third of the bony wall showed bone apposition, in the apical two-thirds, inactivity or slight bone resorption could be seen. Areas of root resorption which were limited to the apical third, had undergone repair by cellular cementum.

Inter-radicular area: The tooth surface and the alveolar crest were separated by a zone of vital connective tissue, the collagen fibers of which were not oriented in any particular direction. The surface of the bifurcation showed repair of previous resorption areas (Fig. 16). The repair was in part anatomic and in part, functional, and had occurred with cellular cementum. The crest of the inter-radicular septum also showed new bone formation which was separated from the old by an irregular reversal line. The width of the new bone was

Fig. 14. Three-month experiment. Buccolingual section through first premolar.

Fig. 15. Three-month experiment. (Area a from Fig. 14)—Note repair of resorbed areas on the alveolar wall. The reversal line (a), indicates the boundary between old and new bone.

greatest on the palatal side of the crest. This was expected because this side was the side of pressure.

The palatal side of the buccal root showed a normal and even width of the periodontal membrane (Figs. 14 & 16). The surface of the socket was irregular but it was evident that resorption had ceased and the bone activity was either at rest or slow bone formation had begun. The areas of root resorption had undergone repair.

On the buccal side of the palatal root, the periodontal membrane appeared normal, and the resorption areas on the root surface as well as the wall of the socket had been repaired by cellular cementum and bundle bone respectively (Figs. 14 & 16).

SIX MONTHS (MONKEY #1)

Six months after the cementing of the inlay, the tooth had moved buccally out of trauma and the opposing teeth on closure were in contact clinically. No changes were noticeable in the gingiva of the experimental and the control sides. The bottom of the sulcus was on the enamel; there was no pocket formation and the few inflammatory cells seen in the free and attached gingiva were not in excess of those seen on the control side.

The microscopic changes in the tooth and its supporting structures at six months postoperatively were similar to those seen at three months. However, the repair process was more complete (Figs. 17 & 18). Wherever, due to pressure, bone had resorbed, new bone was laid down and it was of the bundle bone type, i.e., it gave attachment to the periodontal fibers. The areas

Fig. 16. Three-month experiment. Inter-radicula area. Note repair of the resorption areas in the bifurcation.

of resorption on the surface of the root were all repaired by cellular cementum (Fig. 19). The periodontal membrane showed dense collagen bundles which extended from the bone to cementum and the width of the periodontal membrane was uniform.

The pulp chamber showed a thick layer of irregular dentin. This area corresponded to the pulpal and axial walls of the cavity (Fig. 17).

Fig. 17. Six-month experiment. Buccolingual section through the first premolar area.

DISCUSSION

I. *Occlusal trauma and periodontal disease:* In the present investigation, where teeth were put under excessive occlusal forces for a period ranging from three days to six months, there were no clinical or histological signs of pocket formation, gingival recession, cleft formation, or numerous other lesions ascribed to occlusal trauma. Although the gingival papillae of the experimental teeth showed inflammatory cells, these were in no way more than on the control side. Indeed, it is well known that the presence of some inflammatory cells in the gingiva is "normal." The bottom of the sulcus in all instances was on the enamel. The deepest point of the epithelial attachment in the two younger monkeys was on the cementoenamel junction, while in the two older ones, it was slightly apical to this area. This, however, was not pathological, for a similar relationship could be seen on the control side and is consistent with the normal physiologic

Fig. 18. Six-month experiment. Photomicrogram of the inter-radicular area. Note repair of the areas of root resorption and new bone formation.

passive eruption of teeth.[3] The teeth of the older monkeys showed other signs of age, e.g., their roots were covered by a much thicker layer of cementum. Compare Figs. 1, 6, 8, 14, 16, 18.

In one previous experimental investigation, however, it was shown in a single sheep that occlusal stresses produced by cementing a high crown on an incisor led to pocket formation on the side of tension.[25] It was reported that the experimental tooth showed an abnormal amount of calculus. The deduction has been drawn from the experiment that occlusal trauma causes both pocket formation and deposition of calculus. It seems more logical, however, that the gold crown and not the occlusal trauma was the stimulating factor in the formation of calculus. Once the calculus started to form, the deepening of the crevice resulted. It is also of interest to note that the deepening of the crevice occurred only on the lingual side, the side where one would expect calculus formation. This experiment has been cited again and again as the scientific basis for the belief that occlusal trauma causes periodontal disease. What it really shows, however, is that a rough area on the lingual of a lower anterior tooth would stimulate the deposition of calculus. The sheep has no upper incisors which makes a poor experimental arrangement.

The second report which has claimed a direct relationship between inflammatory periodontal disease and occlusal trauma is that of Stones.[27] High fillings were placed on three adjoining posterior teeth in seven monkeys. The animals were sacrificed 10-43 weeks later. In three of the monkeys, 13-28 weeks postoperatively, the apical end of the epithelial attachment was on the cementum and inflammatory cells were seen in the gingival connective tissue. Although no pocket formation was observed, it was assumed that the presence of the epithelial attachment on the cementum and presence of inflammatory cells in the gingival connective tissue indicated "histologic signs of definite periodontal disease." It was further assumed that these changes forecast a vertical type of pocket formation. A closer study of the illustrations,[27] however, reveals that the tissue changes here described are physiologic changes which occur during passive eruption and that this experiment did not produce "histologic signs of definite periodontal disease." For example, Figure 4 in the original article is intended to show the control sides where, according to the author, the epithelial attachment does not extend along the cementum. It is, however, obvious that the

cementum extends for some distance coronal to the lowest point of the epithelial attachment. The empty space between the uncovered cementum and epithelium is, therefore, an artefact. A similar picture is seen in Figure 9 which is another control. It is thus clear that the controls as well as the experimental first molars showed the presence of the epithelial attachment on the cementum. This is neither unexpected nor pathological. It is well established that in passive eruption, the epithelial attachment migrates along the cementum surface and the longer the tooth has been in the oral cavity, the farther apically does the epithelium migrate.[36] It has been demonstrated by Ritchey and Orban that the alveolar crest follows the cementoenamel junction and this is in relation with the position of the epithelial attachment. In Stones'[27] case, the cementoenamel junction of the supposedly traumatized tooth is on a lower level than the mesially located tooth which explains the difference between the location of the epithelial attachment within the same interdental space.

In Figure 8c, Stones[27] has shown how, in association with a tooth under trauma, a pocket developed. It is clear from the illustration, however, that it does not demonstrate a pocket, but merely an artefact. The epithelial attachment here has separated from the surface of the cementum. No epithelium can be seen on the cementum surface apical to the so-called "pocket."

The presence of inflammatory cells in the gingiva is considered as a definite sign of periodontal disease by Stones.[27] Whereas some inflammatory cells in relation to the bottom of the sulcus are normal, the excessive inflammatory changes seen in some of the illustrations are undoubtedly abnormal. Case #2 of Stones' and Figures 7, 8a, 8b, 8c show such changes. Figures 7, 8b, 8c show, however, that the cause of this gingivitis was not trauma, but the packing of food between the first and second molars.

The relationship between occlusal trauma and periodontal tissues seems to be as follows: The excessive occlusal forces on a tooth can act primarily in five directions—mesial, distal, buccal, lingual and apical. If the excessive forces are directed distally or mesially, and if the adjoining teeth are present, these forces would have no harmful effect on the supporting tissues.[37] In such a case, the adjoining teeth "splint" the tooth under trauma. If, on the other hand, the adjoining tooth is absent, then the occlusal force shall produce the same changes in the periodontal membrane and the bone as are seen in the present investigation. That this, indeed, is the case, has been shown in human jaws.[5,9]

Fig. 19. Six-month experiment. Note repair of resorbed area on the root surface by cellular cementum.

If the excessive force acts in a buccal or lingual direction, but only in *one* direc-

tion, the tooth will attempt to move out of trauma. Its supporting tissues will show changes similar to those seen in the present study and after it has moved out of trauma, repair will ensue.

If an excessive force acts in both buccal and lingual direction, the tooth will show effects of pressure on both sides of the periodontal membrane and will progressively loosen. It loosens because the reparative processes have no opportunity to start. (Such experiments are in progress.)

Excessive forces acting mainly in the apical direction, unless catastrophic, should produce no, or only minor changes in the supporting structures. This is because the fibers of the periodontal ligament by their orientation are best suited to withstand these forces.

It must be understood, however, that the above-described effects refer only to primary occlusal trauma. In secondary occlusal trauma, i.e., where a tooth, due to pre-existing periodontal disease comes under trauma, the changes can be far more complex.

II. *Histologic changes in excessive occlusal stresses:* In this experiment and in a number of previous investigations,[5, 7, 8, 9, 11, 17] it has been shown that excessive occlusal stresses lead to thrombosis and hemorrhage, destruction of bone and necrosis of collagen fibers on the side of pressure, while on the side of tension, new bone formation occurs. In the present study, it is further demonstrated that thrombosis of blood vessels may occur on the side of tension as well. In addition to these changes, it has been shown in previous studies that whereas cementum is more resistant than bone, in some cases, on the side of pressure, it does undergo resorption. In the present study, cementum resorption was seen on pressure sides as well as on the sides of tension and it occurred in all animals. It was most marked at three weeks which at three and six months was completely repaired.

It is of interest to note that in occlusal trauma, although tissue necrosis can be seen in the periodontal membrane, inflammation, as evidenced by migration of cells and exudation, does not occur. It is, however, known that the ligaments and tendons, when severed, heal without inflammation.[38] Since the periodontal membrane is in reality a ligament, when damaged, it is also repaired without inflammation. The mode of repair of the damaged periodontal structures, periodontal membrane, bone, cementum, is such that the destroyed tissue is replaced by the type of tissue lost. That is, resorbed cementum and bone are replaced by cementum and bone respectively and the collagen of the periodontal ligament is replaced by dense collagen. This, in reality, is therefore regeneration.

Because of these important differences between the tissue reaction as seen in occlusal trauma and that seen in true inflammatory diseases of the dental supporting tissues, one should designate "traumatism" as a separate entity. This distinction is necessary, particularly in view of the therapeutic implications. Whereas, in gingivitis and periodontitis, the rationale of treatment is the removal of the source of irritation, namely the pocket or the calculus, in cases of occlusal trauma, the primary therapeutic measure is the elimination of trauma. Whereas it is understood that occlusal trauma cannot cause inflammatory diseases of the dental supporting tissues, it must be remembered that periodontal diseases may lead to occlusal trauma. This can occur by two means: 1. By a destruction of the supporting tissues so that normal occlusal forces are "traumatic" for the involved tooth and 2. By a migration of the tooth, as, for example, in periodontosis, into a place where it is subjected to occlusal trauma. It is obvious, therefore, that in these instances, in addition to other therapeutic measures, the affected tooth should be relieved from these traumatic forces.

SUMMARY AND CONCLUSIONS

To study the relationship between oc-

clusal trauma and periodontal disease, teeth in four monkeys were placed in trauma. To accomplish this, MOD inlays were cemented in the upper first premolars in such a manner that these teeth were subjected to excessive occlusal stresses in buccal direction. The monkeys were sacrificed and profused with 10% formalin at three days, three weeks, three months and six months postoperatively. The jaws were decalcified and embedded in celloidin. The upper premolar area was sectioned serially in a buccolingual direction and the corresponding region of the lower jaw was sectioned in a mesiolingual direction and stained mainly with hematoxylin and eosin. At the start of the experiment and at frequent intervals during its course, Kodachromes and roentgenograms of the experimental as well as the control sides were taken to determine objectively any changes in the position of the tooth or the color and texture of the gingival tissues.

THE FINDINGS

1. Three days after cementing the inlay, the tooth moved buccally so that the space between the opposing teeth in the rest of the arch was reduced. At three weeks, the movement of the tooth had progressed far enough that all the teeth were in occlusion.

2. Three days after the tooth was put under trauma, the pressure areas showed necrosis of periodontal fibers, thrombosis, and resorption of bone while the tension areas showed widening of the periodontal space and thrombosis.

3. At three weeks, the changes were similar, but far more pronounced than at three days. The alveolar wall in the tension areas showed beginning bone formation. The most striking change, however, was the presence of numerous areas of root resorption on both the tension and pressure sides.

4. Three and six months stages were characterized by repair. The periodontal membrane was of normal width, necrotic bone and connective tissue had been replaced by new bone and dense collagen bundles and the resorbed areas on the root had undergone repair. This repair was predominantly anatomic.

5. Clinically, the gingiva in none of the monkeys showed *lesions comparable to gingivitis or periodontitis.*

6. Pocket formation was not seen in any instance. Inflammatory cells were seen in the gingiva but these were not in excess of those seen on the control side.

From this experiment it was concluded that:

1. Occlusal trauma, contrary to various claims, does not produce gingivitis, periodontitis, pockets, pulp stones, recession, festoons, clefts, erosion or caries.

2. Changes seen in occlusal trauma are specific and in their extreme consist of necrosis of periodontal tissue, resorption of bone, thrombosis and root resorption on the sides of pressure and widening of the periodontal membrane, thrombosis and bone formation on the sides of tension. In some tension areas, root resorption also occurred.

3. The changes seen in occlusal trauma are reversible.

Dr. Bhaskar is in the Dept. of Oral Pathology, University of Illinois College of Dentistry, Chicago, Illinois.

Dr. Orban is associated with the Dept. of Periodontics, Loyola University School of Dentistry, Chicago, Illinois and the Colorado Dental Foundation, Colorado Springs, Colorado.

BIBLIOGRAPHY

1. Karolyi, M. Beobachtungen über Pyorrhea Alveolaris. Oest. ung. Vierteljschr Zahnheilk 17:279, 1901.

2. Robinson, H. B. G. Discussion: Relation of Histology to Crown and Bridgework. J. Dent. Ed. 5:328, 1941.

3. Box, H. K. Traumatic Occlusion and Traumotogenic Occlusion. Oral Health 20:642, 1930.

4. Glickman, I. Clinical Periodontology. W. B. Saunders Co., Philadelphia, 1953.

5. Orban, B. Tissue Changes in Traumatic Occlusion. J.A.D.A. 15:2090, 1928.

6. McMillan, H. W. The Case Against Traumatic Occlusion. J.A.D.A. 17:1996, 1930.

7. Gottlieb, B., and Orban, B. Tissue Changes in Experimental Traumatic Occlusion with Special Reference to Age and Constitution. J. D. Res. 11:505, 1931.

8. Gottlieb, B., and Orban, B. Die Veranderungen der Gewebe bei ubermassiger Beanspruchung der Zahne, Thieme, Leipzig, 1931.

9. Orban, B., and Weinmann, J. P. Signs of Traumatic Occlusion in Average Human Jaws. J. Dent. Res. 13:216, 1933.

10. Boyle, Paul E. Experimental Production of Diffuse Alveolar Bone Atrophy Type of Periodontal Disease by Diets Deficient in Ascorbic Acid (Vit. C). J.A.D.A. 24:1768, 1937.

11. Orban, B. Traumatic Occlusion and Gum Inflammation. J. Period. 10:39, 1939.

12. Merritt, A. H. Traumatic Occlusion: Cause or Effect in Periodontal Disease? N. Y. State Dent. Journal 15:507, 1949.

13. Bodecker, C. F. Discussion in: Traumatic Occlusion—Cause or Effect in Periodontal Disease? N. Y. State Dental Journal 15:507, 1949.

14. Beube, F. K. Periodontology. MacMillan Co., New York, 1953.

15. Fish, E. W. Paradontal Disease—2nd Ed. J. B. Lippincott Co., Philadelphia, 1953, p. 37.

16. Glickman, I., and Weiss, L. Changes in Periodontium of Dogs Associated with Increased Occlusal Stress 26:14, 1955.

17. Macapanpan, L. C., and Weinmann, J. P. The Influence of Injury to the Periodontal Membrane on the Spread of Gingival Inflammation. J. Dent. Res. 33:263, 1954.

18. Robinson, H. B. G. Chapter on Periodontal Disease in "Oral Hygiene" by Bunting, R. W. Lea and Febiger, Philadelphia, 1954, p. 177.

19. Stillman, P. R. Traumatic Occlusion. J. of National Dental Association 6:691, 1919.

20. Box, H. K. Studies in Periodontal Pathology. Bulletin No. 7, Canadian Dental Research Foundation, 1924.

21. Box, H. K. Treatment of the Periodontal Pocket. Bulletin No. 10 Canadian Dental Research Foundation, 1928.

22. Gracey, C. H. The Clinical Significance of Traumatic Occlusion. J.A.D.A. 17:2009, 1930.

23. McCall, J. O. Principles of Traumatic Occlusion and their Application to the Field of Operative Dentistry. J.A.D.A. 17:609, 1930.

24. Sorrin, S. Traumatic Occlusion—Its Detection and Correction. Dent. Digest 40:170, 1934.

25. Box, H. K. Experimental Traumatogenic Occlusion in Sheep. Oral Health 25:9, 1935.

26. Balfe, R.; Carranza, R., and Erausquin, R. The Periodontal Structures in Case Number 8. Revista Odontologica, Buenos Aires, July, 1938.

27. Stones, H. H. An Experimental Investigation into the Association of Traumatic Occlusion with Parodontal Disease. Royal Society of Medical Practice 31:479, 1938.

28. Box, H. K. Discussion in: Traumatic Occlusion—Cause or Effect in Periodontal Disease. N. Y. State Dental Journal 15:507, 1949.

29. McCall, J. O. Discussion in: Traumatic Occlusion: Cause or Effect in Periodontal Disease? N. Y. State Dental Journal 15:507, 1949.

30. Kerr, D. A. "Traumatic Occlusion"—in R. W. Bunting's "Oral Hygiene and Prœventive Dentistry." Lea and Febiger, Philadelphia, 1950.

31. Bunting, R. W. Oral Hygiene and Preventive Dentistry. Lea and Febiger, Philadelphia, 1954, p. 18.

32. Thoma, K. H. Oral Pathology. C. V. Mosby Co., St. Louis, Mo., 1954.

33. Ostrom, C. A.; Skillen, W. G., Fosdick, L. S. "Gingival Glycogen Concentration in Experimental Occlusal Trauma." J. Dent. Res. 29:55, 1950.

34. Glickman, I., and Weiss, L. A. Role of Trauma from Occlusion in Initiation of Periodontal Pocket. J. Period. 26:14, 1955.

35. Waerhaug, Jens. Pathogenesis of Pocket Formation in Traumatic Occlusion. J. Period. 26:107, 1955.

36. Orban, B. Oral Histology and Embryology—3rd Ed. C. V. Mosby Co., St. Louis, Mo., 1953.

37. Ritchey, B., and Orban, B. The Crests of the Interdental Alveolar Septa. J. Period. 24:75, 1953.

38. Willis, R. A. The Principles of Pathology. The C. V. Mosby Co., St. Louis, Mo., 1950.

Healing of Naturally Occurring Periodontal Pockets About Vital Human Teeth

by MELVIN L. MORRIS, B.S., M.A., D.D.S.,* *New York, N. Y.*

DESCRIPTIONS of healing or reattachment of human periodontal pockets have been legion. The procedures employed have attempted to remove foreign matter and outer cemental layers from the root surface, and epithelium from the soft tissue wall. They have been based on the theory that the resultant bleeding would produce a clot in the pocket space which would be replaced successively by granulation tissue and a new connective tissue union with the root. The evidence produced by these studies have, in the main, been changes in pocket depths or the radiographic picture.

Changes in pocket depth do not reveal the type of connection, which may be epithelial, connective tissue, or simply a tight apposition of tissue.

Dental radiographs reveal root and bone, but not soft tissue attachment. Since there is no constant relationship between the relative heights of bone and attachment,[4, 5, 7] the mere demonstration of bone growth becomes inconclusive evidence of reattachment. The use of radiopaque points before and after treatment[6] does add some credence to the accompanying radiographs.

Histologic evidence, correlated with clinical data, must be the ultimate proof. Such studies have been scarce, only eight human tooth and tissue biopsies being available in the literature for inspection.[2, 3, 13, 15] All but one[15] have shown both epithelial and connective tissue union in restricted areas. Such sections, without root landmarks,[5] do not indicate whether they are coronal to, adjacent to, or apical to the level of the original epithelial attachment.

The one exception[15] mentioned above was Waerhaug's study, in which root nicks were employed as landmarks. Five tooth and tissue biopsies were obtained. The one illustrated showed epithelial lining down to the root nick, the remaining four specimens reportedly presenting the same status. Simple extractions were then performed on thirty-two teeth after attempting reattachment and the relative positions of root nicks and clinging epithelium and connective tissue was observed. Slight amounts of healing were seen only in the four cases in which the pockets had been artificially deepened.

There seemed to be a need for more tooth and tissue sections correlated with clinical data. Furthermore, it was felt[8] that complete cemental removal would increase the chances for healing by exposing vital dentin. The present report attempts to fulfill these conditions.

PROCEDURE

The study was conducted on vital upper anterior teeth, previously condemned for prosthetic or periodontal reasons. Labial periodontal pockets were the experimental sites chosen for ease of access, simplicity of procedure, minimal patient discomfort, and absence of deformity following biopsy.[9]

Root surfaces were thoroughly curetted one week previous to the experimental routine in order to produce gingivae that were firm, pink and stippled. The procedure was then started with the grinding of tooth nicks with a #2 wheel bur, varying their placement somewhat in the different cases. The first nick was made on the labial enamel (Fig. 1, N_1) and the second at pocket depth (Fig. 1, N_3).

The pockets were re-curetted and cemental removal attempted. Pocket and attachment epithelium were eliminated with a standardized technique of curettage,

*Assistant Clinical Professor of Dentistry, Division of Periodontology, Columbia University, School of Dental and Oral Surgery.

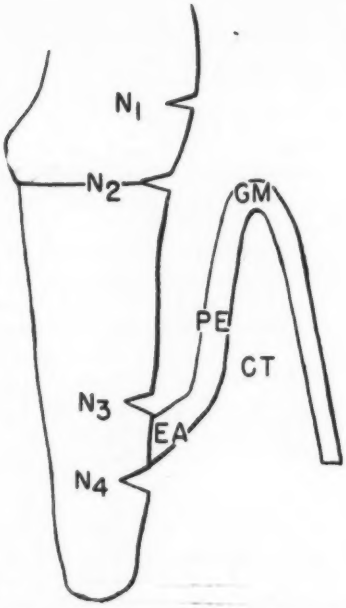


Fig. 1. The Location of Hard Tissue Landmarks. GM—Gingival Margin, PE—Pocket epithelium, EA—Epithelial attachment, N_1—Nick on labial enamel as fixed point for measurements, N_2—Nick at level of gingival margin, N_3—Nick at bottom of preoperative pocket, N_4—Nick at most apical point immediately after removal of epithelial attachment.

Fig. 2.—Biopsy Technique. P.P.—Area covered by original pathological pocket. L.I.—Line of incision isolating soft tissue to be removed with tooth.

excision of gingival rim and irrigation.[10] A nick was placed in some cases at the level of the gingival margin (Fig. 1, N_2) immediately following removal of the gingival rim. Another one was also placed as far apical as possible, after removal of the epithelial attachment (Fig. 1, N_4). Fresh bleeding was provoked and sutures were drawn through the adjacent papillae, encircling the teeth, holding tissue against root. The operated areas were protected with gauze for ten minutes.

Measurements were recorded to fix the positions of pocket bottoms and gingival margins pre and post-operatively.

Ten cases were operated on with a total of fifteen pockets. Teeth and labial tissue (Fig. 2) were removed with a method[9] adapted from Box,[3] after healing intervals ranging from 42 to 140 days. The extracted masses were then fixed in Bouins solutions, decalcified, imbedded in paraffin, sectioned, and stained with hematoxylin and eosin.

RESULTS

A. *Clinical*

Utilizing the figures in Table 1, the results may be summarized as follows:

a. Level of Pocket Bottom

1. Nine showed no change in position.
2. Two had advanced one half millimeter coronally.
3. One had advanced one millimeter coronally.
4. Two receded one millimeter apically.
5. One receded two millimeters apically.

b. Level of Gingival Margin

1. Two showed no change in position.
2. Six receded one half millimeter.
3. Five receded one millimeter.
4. One receded two and one half millimeters.
5. One receded three and one half millimeters.

B. *Histological*

Case #1 (Fig. 3) is a 70 day specimen, with the root surface curetted to remove cementum and in which a nick was made (N_2) at the gingival margin. The gingival margin remains adjacent to the nick.

TABLE 1.
Millimeter Measurements

Case #	Tooth	Days of Healing	Pre-operative		Post-operative			Clinical Reattachment	Gingival Recession
			Nick to Pocket Depth	Nick to Gingival Margin	Nick to Gingival Margin Immed. Post-Op	Nick to Pocket Depth	Nick to Gingival Margin at time of Biopsy		
1	1/	70	7.0	2.5	3.0	9.0	3.0	-2	0
2	3/	140	3.5	0.5	1.0	3.0	2.0	0.5	1
3	/1	70	3.5	0	0.5	2.5	0.5	1.0	0
4	1/	27	7.0	1.0	1.5	7.0	4.0	0	2.5
5	1/	69	7.0	1.5	1.5	7.0	5.0	0	3.5
6	/1	24	3.0	0	0.5	3.0	0	0	0.5
7	/1	42	3.5	0.5	1.0	3.5	2.0	0	1
8	/2	49	4.0	1.0	1.5	4.0	1.0	0	0.5
	/3		4.0	1.0	1.5	3.5	0.5	0.5	1.0
	/4		3.0	1.0	1.5	3.0	1.0	0	0.5
9	2/	35	12.0	3.0	4.0	12.0	3.0	0	1.0
10	1/	28	4.0	2.0	2.0	5.0	3.0	1.0	1.0
	2/		6.0	1.5	2.5	6.0	2.0	0	0.5
	/1		5.0	2.0	2.0	6.0	2.5	1.0	0.5
	/2		5.0	0.5	1.0	5.0	1.5	0	0.5

Inspection shows that the original cementum (C) actually remains, as does the deep pocket.

Case #2 (Fig. 4) is a 140 day specimen, with cementum removed by a bur and a nick (N_3) made at the depth of the pocket pre-operatively. Connective tissue attachment (CT) to the root begins at the apical end of this terminal nick.

Case #3 (Fig. 5) is a 70 day specimen, with the deeper layers of dentin exposed. A nick (N_4) was made following removal of epithelial lining and attachment. Old cementum (C) remains near the depth of the original pocket. New cementum (NC) is present in the nick (N_4) against dentin (D). Figure 5A, a higher magnification of N_4 shows the new cellular cementum (NC) growing on dentin (D) and old cementum (C). A is artefact separating connective tissue (CT) from the root.

Case #7 (Fig. 6) is a 42 day specimen from which cementum was to be removed from the coronal half of the pocket, and nicks made as far apical as possible before (N_3) and after (N_4) removal of epithelial attachment. Actually, all cementum was removed up to and including nick N_3. A is a shrinking artefact. Healing has occurred in N_4. AB is alveolar bone. Figure 6A is a higher magnification of N_4. Connective tissue (CT) is joined with the dentin (D) at the new cementum (NC).

Case #8 (Fig. 7) is a 49 day specimen from which cementum was to be removed from the coronal half of the pocket, and nicks made as far apical as possible before (N_3) and after (N_4) removal of epithelial attachment. Old cementum (C) may be seen. N_3 remains in the pocket. Connective tissue attachment (CT) may be seen in the area between N_3 and in N_4 and in N_4 itself. Figure 7A, a higher magnification of N_4, shows the union of connective tissue (CT) in N_4 and a bit coronal to this point. Cases #2, 3, 7 and 8 exhibit considerable shrinkage of the free gingival tissue in the processing of the sections.

DISCUSSION

A. *Clinical*

Pocket measurements are subject to certain inherent inaccuracies. Where the markings on the probe are a millimeter apart an error of at least $\pm$ 0.5 mm. must be allowed.[11] In addition, there are other

Fig. 3. Deep pocket remaining after reattachment procedure. N_3 indicates original position of gingival margin, which has not changed in this case. C is old cementum.

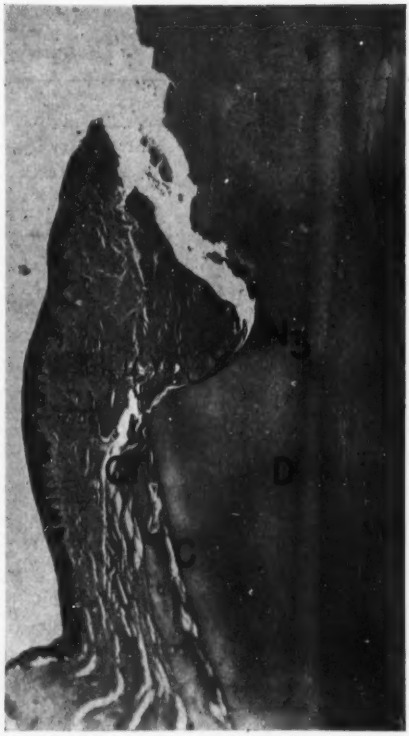


Fig. 4. Connective tissue attachment (CT) starting near depth of original pocket (N_3). D is dentin, and C is old cementum.

sources such as parallax, deviation of probe from the vertical, thickness of probe, and tight adaption rather than actual union of soft tissue to root. Within the range of errors discussed above, it may be said that pocket bottoms have not been advanced in a coronal direction by the experimental procedure. Several showed a very slight apical movement, a surgical risk anticipated by Ritchey and Orban.[12]

These results disagree with the clinical measurements of Schaffer and Zander[13] who reported three out of eight pockets showing at least two millimeters of coronal advance of the pocket bottom, and Shapiro[14] who made similar claims for thirty out of thirty-six pockets. One possible explanation for the varying results might be the location of the pockets since all eight of Schaffer and Zander's and thirty-two of thirty-six of Shapiro's were proximal in location whereas all in the present study were on the labial surface. It has been an oft expressed clinical observation that healing is more likely to occur in infrabony pockets, which are most usually found on proximal or palatal root surfaces. Whether location is important or not is still a matter for conjecture. It may be noted that a study of the healing of surgically detached periodontal tissues[9] was conducted in the same locations and in exactly the same manner as in the present paper. The uniform healing recorded in that previous experiment was not adversely affected by location. It is our opinion that the patho-

Fig. 5. Formation of new cementum (NC), at the end of the original epithelial attachment (N_4), on dentin (D) and old cementum (C).

Fig. 5A. Inset of Fig. 5 magnified. The thick layer of new cellular cementum (NC) is seen with the connective tissue of the periodontal membrane (CT) clinging to it. A is shrinkage artefact.

Fig. 6. Shrunken gingival tissue not attached at depth of old pocket (N_3) but connected to root more apically down to bottom of original epithelial attachment (N_4). A is shrinkage artefact, AB alveolar bone and C old cementum.

logical tissue changes involved in pocket formation rather than location are responsible for lack of healing.

The average gingival recession of slightly less than one millimeter is similar to that noted in the healing of surgically created pockets.[9] It is probably due to the natural contraction of healing connective tissue which occurs whether or not union with the root is accomplished.

B. *Histological*

The positions of the nicks indicate that healing did not occur coronal to the bottom of the original pocket, whether the root surface was cementum or dentin. Somewhere near the apical end of the

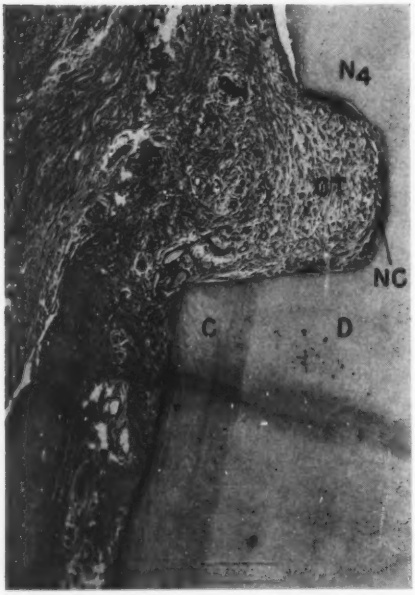


Fig. 6A. Inset of Fig. 6 magnified. Healing noted with formation of new cementum (NC) on dentin (D) and old cementum (C).

epithelial attachment the reaction changed and healing did occur with formation of cementum, against either dentin or old cementum, this being the same phenomenon observed with surgically detached tissues.[9] Confirming the work of Waerhaug,[15] there was reunion only where a recent union was artificially disrupted. Such a distinction can be made only in the presence of root landmarks, unless the healing covers a convincingly large part of the root. None of the studies without landmarks[2, 3, 13] satisfy this criterion.

There are certain important differences between artificially induced and naturally occurring periodontal pockets besides manner of origin (Fig. 8). Naturally occurring periodontal pockets have epithelial linings and a cemental wall cut off from its main source of nutrition.[8] With all the cementocytes in this wall dead the pocket cementum may be considered non-vital.[7] Further differences have been reported by Bass[1] in the form of "pathologic granules" in the dentin close to the dentino-cemental junc-

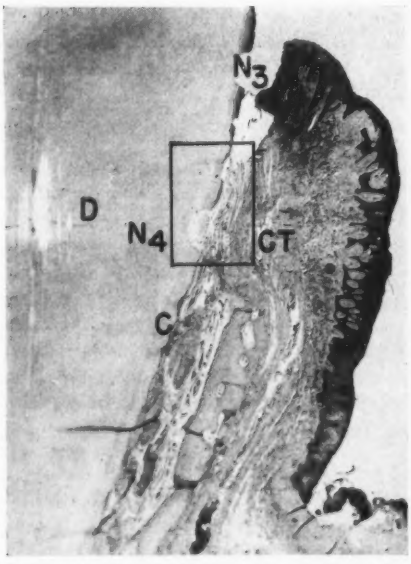


Fig. 7. Connective tissue (CT) attachment begins below original pocket depth (N_3), down to bottom of original epithelial attachment (N_4) and is on old cementum (C) and dentin (D).

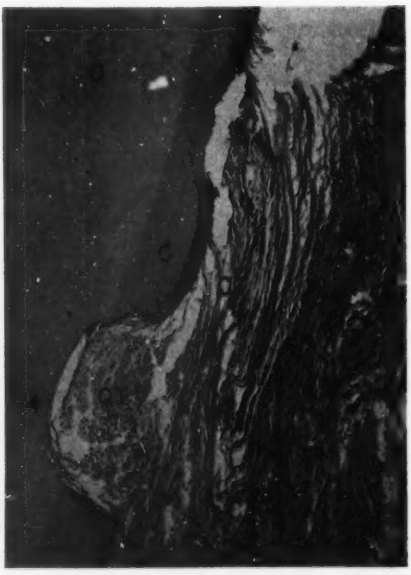


Fig. 7A. Inset of Fig. 7 magnified. The connective tissue attachment (CT) is at N_4 and slightly coronal to that point.

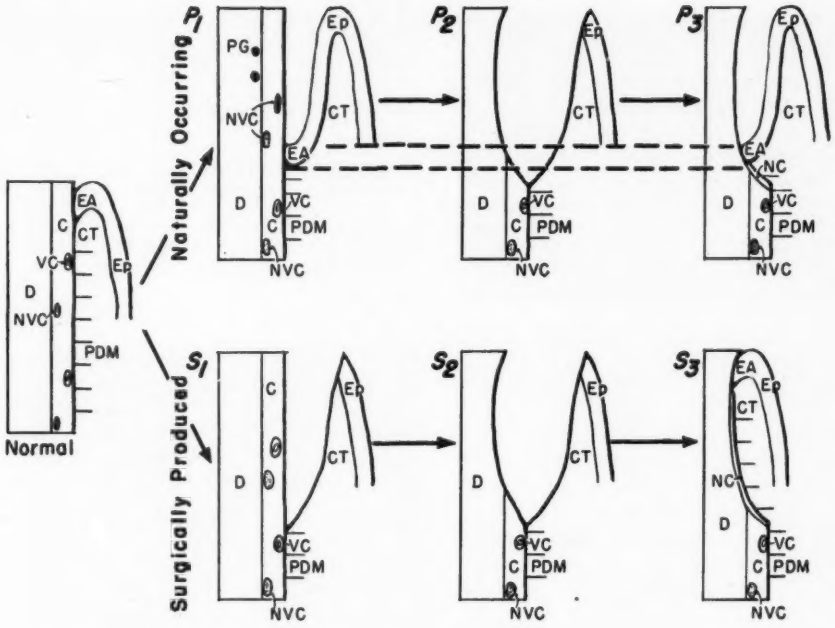


Fig. 8. The formation and healing of surgical and pathological periodontal pockets in vital teeth. Although the histological appearance of P_2 and S_2 are identical, the healing reactions as seen at P_3 and S_3 are quite different. Surgical pockets heal completely whereas pathological pockets heal only at the area of the epithelial attachment (EA) and apical to that location. D—Dentin, C—Cementum, VC—Vital Cementocytes, NVC—Non-vital Cementocytes, EA—Epithelial attachment, PDM—Periodontal Membrane, CT—Connective Tissue, EP—Epithelium, PG—Pathological Granules of Bass, NC—New Cementum.

tion adjacent to naturally occurring pockets. The removal of pocket and attachment epithelium, cementum, and some dentin produces a picture histologically identical with the surgically produced pocket (Fig. 8). The healing of the latter and lack of healing of the former suggests that the aforementioned resemblance is probably only a morphological one. More subtle physiological differences must exist to account for the differing healing reactions.

The author wishes to express his appreciation for the advice and constructive criticisms by Drs. Barnet Levy, Herbert Bartelstone, Frank E. Beube and Solon Ellison; for the preparation of histological material by Miss Mary Minasian: and for the aid in obtaining biopsies by Miss Elise Boyd and Drs. Salvatore Gulli, Stanley Heller, Samuel Touroff and Boas Schattan.

SUMMARY

Clinical and histological data appeared to show that removal of epithelial lining and attachment did not produce healing against curetted cementum or exposed dentin in naturally occurring periodontal pockets about vital human teeth.

BIBLIOGRAPHY

1. Bass, C. C.: A Previously Undescribed Demonstrable Pathologic Condition in Exposed Cementum and the Underlying Dentine. Oral Surg., Oral Med., Oral Path., 4:641-651, 1951.

2. Beube, F. E.: A Radiographic and Histologic Study on Reattachment. J. Perio. 23:158-164, 1952.

3. Box, H. K.: Treatment of the Periodontal Pocket. Toronto, University of Toronto Press, 1928.

4. Goldman, H. M.: Relationship of the Gingival Crevice and the Alveolar Crest. J. D. Res. 21, 561-564, 1942.

5. Gottlieb, B.: The New Concept of Periodontoclasia. J. Perio. 17:7-23, 1946.

6. Hirschfeld, L.: A Calibrated Silver Point for Periodontal Diagnosis and Recording. J. Perio., 24:94-98, 1953.

7. Kronfeld, R.: Histopathology of the Teeth and Their Surrounding Structures. Phila., Lea and Febiger, 1945.

8. Morris, M. L.: Reattachment of the Periodontal Tissue. Oral Surg., Oral Med., Oral Path., 2:1194-1198, 1949.

9. Morris, M. L.: The Reattachment of Human Periodontal Tissues Following Surgical Detachment: A Clinical and Histological Study. J. Perio., 24:220-228, 1953.

10. Morris, M. L.: The Removal of Pocket and Attachment Epithelium in Humans, A Histological Study. J. Perio., 25:7-11, 1954.

11. Ramfjord, S.: Experimental Periodontal Reattachment in Rhesus Monkeys. J. Perio., 22:67-77, 1952.

12. Ritchey, B., and Orban, B.: The Periodontal Pocket. J. Perio., 23:198-212, 1952.

13. Schaffer, E. M., and Zander, H. A.: Histological Evidence of Periodontal Reattachment of Pockets. Paradentologie 3:101-107, 1953.

14. Shapiro, M.: Reattachment in Periodontal Disease. J. Perio. 24:26-31, 1953.

15. Waerhaug, J.: The Gingival Pocket. Odontolgisk Tidskrift, 60, 1952. Supplementum 1, Oslo, Norway.

The Healing of Periodontal Wounds in Dogs

by M. T. JANSEN, M.D.; L. COPPES, D.M.D., AND H. H. W. VERDENIUS, D.M.D.,
Utrecht, Netherlands

INTRODUCTION

THE question whether or not a severed attachment of the tooth can heal, is of obvious importance to all concerned with the treatment of the pathological periodontal pocket. Most workers agree that reattachment does take place provided the oral epithelium does not interfere with it. With two exceptions[1,2] the technics of the investigators have in common the free communication of the experimental wound with the oral cavity. The present report deals with an investigation in which the healing of periodontal lesions under constant contamination of the wound by oral contents has been compared with the repair of similar lesions in which contamination and the ingrowth of oral epithelium could be excluded.

METHODS AND RESULTS

All experiments reported here were performed on six adult dogs which were operated upon (as detailed below) under pentothal-sodium anesthesia. Three dogs lived for two or three weeks, and the remaining animals for two to three months after the operations. After the animals had been sacrificed the heads were perfused with saline, followed by 10% formol solution. The tissue-blocks were decalcified and embedded in paraffin according to Morse[3] and sectioned serially. Staining was routinely done with haematoxylin and eosin.

A

The periodontal lesions which were intended to heal unaffected by contamination and epithelial ingrowth had been caused by burs or discs introduced through the labial alveolar walls. To this end a gingival flap was prepared in the region of the roots of the premolars. The rapidly rotating cutting instruments were cooled with saline. After the required amount of tissue had been removed the gingiva was

sutured carefully. In these cases we thus destroyed part of the alveolar wall and of the periodontal membrane. In addition, more or less deep excavations of the roots were caused (fig. 1*).

After two to three weeks the bloodclot has been cleared away and replaced by connective tissue (fig. 2). This tissue is denser and subject to less histological shrinkage in the bone defect than in the

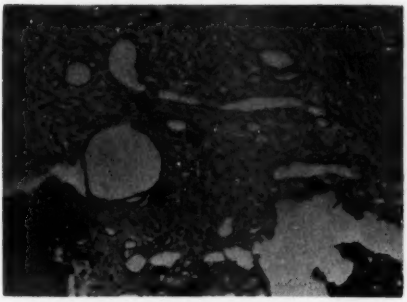


Fig. 2. Higher magnification of figure 1: granulation tissue in alveolar and rootdefects. Magn. 110 x

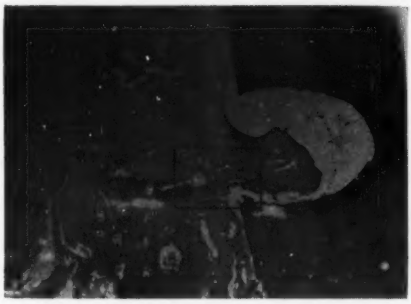


Fig. 1. Root of upper third premolar of dog, 3 weeks after the operation. Magn. 25 x.

root excavation. There are many osteoclasts lying against the bony spicules of the alveolar wall and attacking loose particles of bone, dentin and cementum. No osteoclasts border the wall of the root defect, though occasionally cementum resorption may be found on the undamaged root surface. Osteoblasts are not active at this stage.

After two or three months the gap in the alveolar bone is completely filled. The site of the operation can be ascertained from the slightly different aspect of the newly formed bone. The root defect on the contrary remains clearly discernible, as only the shallowest defects are completely filled up with cementum. Quite often large areas of the deeper excavations are not covered with cementum at all. Notwithstanding this the periodontal membrane has generally assumed the normal breadth throughout (fig. 3). This implies that bony excrescences of the alveolar wall bulge into the larger defects of the root. The outlines of these protrusions often quite accurately follow those of the root

Fig. 3. Root of lower third premolar of dog, 2 months after lesion set by diamond disc. Extensive repair of the defect in the alveolar wall and reconstitution of a periodontal membrane of normal width. Magn. 25 x.

*Figures 1 to 8 inclusive: Injuries of periodontal tissues made by introducing a bur or a diamond disc through the alveolar wall into the periodontal space.

Fig. 4. Higher magnification of figure 3, showing apposition of new cementum and reattachment of newly formed collagen fibers. The new cementum has been partly torn away from the dentin surface. Magn. 110 x.

Fig. 5. Higher magnification of figure 4. Magn. 225 x.

defects, irrespective whether or not these defects are completely lined with cementum.

From the aspect of the border line between cementum and dentin one may conclude that the deposition of new cementum has as often as not been preceded by osteoclastic resorption. Reattachment did occur (figs. 3 to 8 inclusive), but it is of course lacking in those areas where no new cementum had been laid down. No rule could be found governing the distribution of these areas. The connection of the newly formed cementum to the tooth is rather weak as it is often found to be disrupted, either intra vitam or during the preparation of the specimen (figs. 4 and 5).

B

Two sets of lesions were made to imitate more closely the conditions of the pathological periodontal detachment, i.e., the communication of the wound with the oral cavity.

(1) The first category of lesions was brought about by introducing a slender and sharp scalpel through the gingival crevices so as to cause a minimum of destruction except the severance of the periodontal fibers close to the cementum. The approximal aspects of incisors were used for these experiments. Teeth studied immediately after the operation revealed the feasibility of the method, though occasionally small particles of cementum and dentin were scraped off.

After two or three weeks the connective tissue generally shows no sign of inflammation. There is no reattachment, the connective tissue receding somewhat from the cemental surface because of the histological shrinkage. Though the ensuing cleft between the tooth and the periodontium may be occupied by an epithelial downgrowth from the gingiva, in other cases no trace of epithelium may be found in it (figs. 9* and 10). In some instances however the reaction was much stronger. The connective tissue contains infiltrates and the epithelium had proliferated along the root surface and into the connective tissue. Where the thickness of the epithelial downgrowth exceeds 4 or 5 cell-layers, the cells in the middle layers are less distinctly colored. Moreover, the epithelium tends to be torn up in these layers, which seems to confirm the theory of the intra-epithelial origin of the pocket (figs. 11 and 12).

Reattachment of the periodontium to the tooth surface through the deposition of new cementum is found after two or three months (fig. 13). Sometimes the old root surface had apparently been excavated by osteoclasts prior to the formation of new cementum. Small particles of cemen-

*Fig. 9 to 17 inclusive: lesions made by introducing slender knife through gingival crevice into periodontal membrane.

Fig. 6. Root of lower fourth premolar of dog. 2 months after injury set by diamond disc. Newly formed alveolar bone and repair of the periodontal membrane. Magn. 25 x.

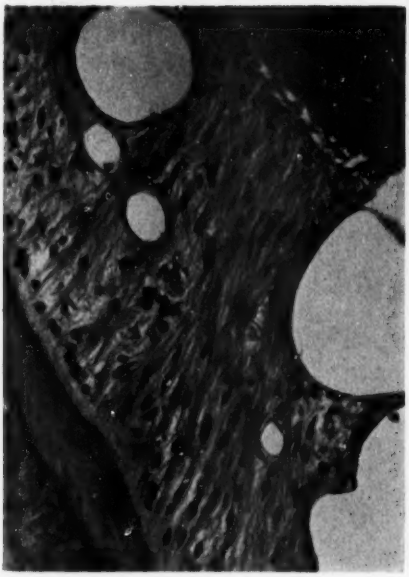


Fig. 7. Higher magnification of figure 6: reattachment. Magn. 225 x.

tum and dentin chipped off the root surface during the operation were retrieved in the periodontium solidly embedded in a mass of bone (figs. 14 to 17 inclusive). They seemed scarcely to have been attacked by osteoclasts.

(2) A much more serious destruction of the paradental tissues was caused by introducing a round bur through the gingival crevice into the periodontal space. This procedure which has been applied to the canines of the dogs used in the other experiments locally destroys the gingiva and the periodontal membrane besides excavating the upper part of the root and damaging the alveolar crest.

The findings, two or three weeks later, reflect the havoc caused. The connective tissue is inflamed, the borderline between it and the epithelium is highly irregular and there is heavy osteoclastic activity. The epithelium has proliferated much more extensively than after the mere cutting

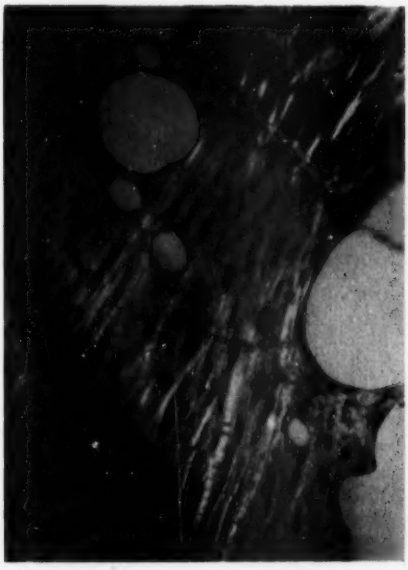


Fig. 8. The same as figure 7. Polarized light. Magn. 225 x.

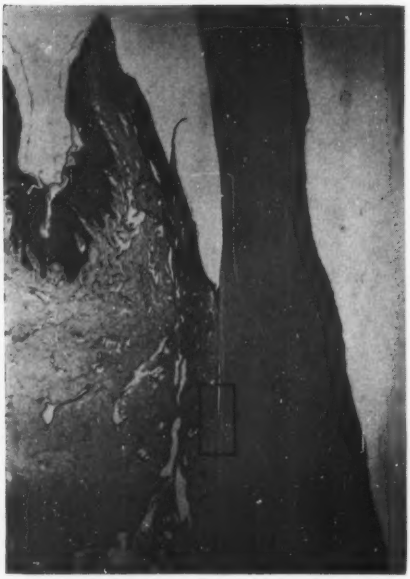


Fig. 9. Upper incisor of dog, 3 weeks after the operation. Crevice between cementum and periodontal membrane. In serial sections no communication with the gingival crevice appeared to exist; in this case the lancet bypassed the epithelial attachment, taking its way through the interdental papilla. Magn. 25 x.

Fig. 11. Lower incisor of dog, 2 months after lesion; proliferation of the epithelium along the cementum. The surrounding connective tissue shows infiltration with inflammatory cells. Rupture in the epithelium: formation of pocket. Magn. 25 x.

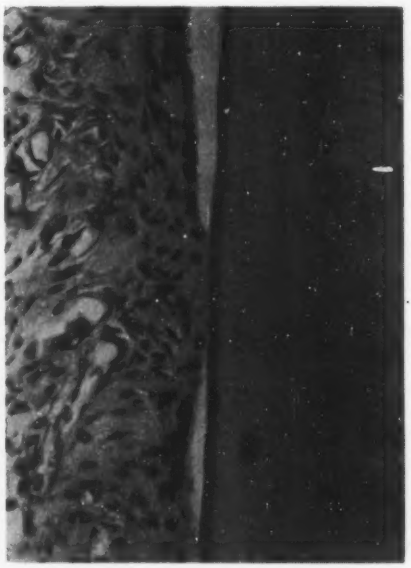


Fig. 10. Higher magnification of figure 9. No reattachment, no epithelial covering. Magn. 225 x.

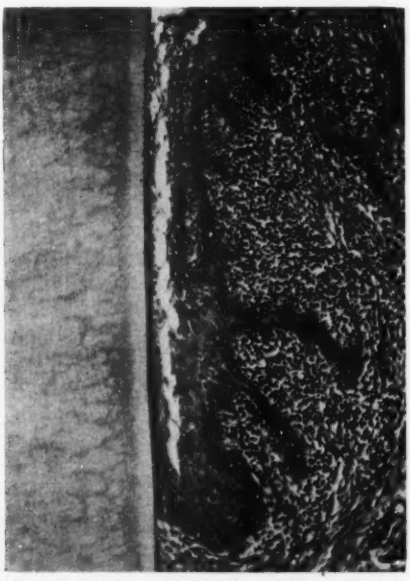


Fig. 12. Higher magnification of figure 11. Note epithelial cells attached to the cementum surface: intraepithelial formation of pocket. Magn. 110 x.

Fig. 13. Lower incisor of dog, 3 months after lesion. Formation of new cementum and reattachment. Magn. 110 x.

of the periodontal fibers. It often covers a large portion of the excavation in the root and it sends pegs and plates into the connective tissue. Osteoclasts lie against the dentin and the cementum of the root and along the inner and outer surfaces of the alveolar wall.

After two or three months the picture has changed radically. Inflammatory signs have been reduced to a slight round-cell infiltration underneath the epithelium. The upper parts of the root defects may be covered with epithelium and the balance with newly formed cementum but this must not be so. In some instances the cementum covers only a small part of the defect though there is no epithelial downgrowth occupying the rest of the surface (figs. 18, 19 and 20).

In these experiments, too, the cementum is easily detached from the dentin whether or not prior to the formation of cementum the dentin had been eroded by osteoclasts.

Osteoblasts and osteoclasts often have remodeled the surface of the alveolar bone so as to make it project into the root defect. The loose particles of cement and dentin have by this time become enclosed in solid bone-like masses, which are occasionally attached to the alveolar wall or to the cementum.

DISCUSSION

Though apparently there can be little doubt that reattachment can take place we should consider some aspects more closely.

If we follow the course of events in those cases where repair went on uninfluenced by contamination from the oral cavity or by the ingrowth of epithelium, a periodontal space of normal breadth is found to be the outcome. Yet large areas of the root defects may be devoid of a cemental covering. As there cannot be any adhesion of connective tissue to the dentinal surface except after the deposition of cementum, we are led to the conclusion that the attachment of the periodontal membrane to the tooth surface is not essential for the establishment of a periodontal space of normal breadth. Though this might indicate that mechanical forces are not involved at all in this particular case of restricted bone formation, one should keep in mind, that the breadth of the periodontal space could conceivably be conditioned by the way the root is pressed against the alveolar wall during the excursions of the tooth rather than by traction through the periodontal fibers. The well known narrowing of the periodontal spaces of inactive teeth points into the same direction.

The frequently observed looseness of the connection between the newly laid down cementum and the underlying dentin seems to be more understandable than the firmness of the natural cemento-dentinal junction. In fact, in the absence of connective tissue fibers running from the one tissue to the other, one wonders why the cementum should not come off the dentin rather easily. Though the adherence of the ce-

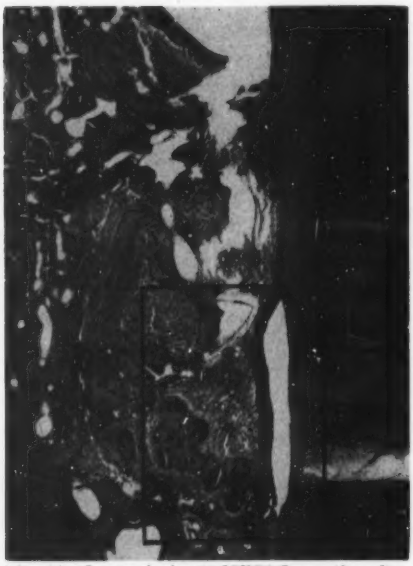


Fig. 14. Lower incisor of dog, 3 months after lesion. Formation of new cementum which has been torn away from the injured dentin surface. Particles of cementum and dentin incorporated in newly formed bone tissue which has grown together with the layer of new cementum. Magn. 25 x.

(The detachment of the soft tissue from the tooth surface in the upper part is due to the preparation of the specimen.)

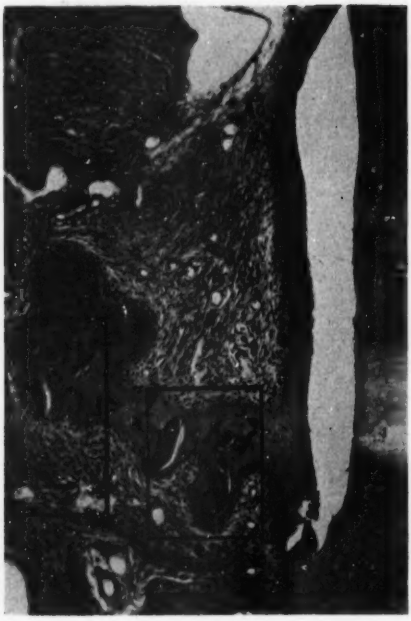


Fig. 15. Higher magnification of figure 14, showing relation of the detached cementum layer and the newly formed bone which envelops particles of cementum and dentin. Magn. 110 x.

Fig. 16. Higher magnification of figure 15 (left part). Collagen fibers entering newly formed bone. Magn. 225 x.

Fig. 17. Higher magnification of figure 15 (right part). Newly formed bone enclosing particles of dentin. Magn. 225 x.

Fig. 18. Root of upper incisor of dog, 2 months after lesion made by burring through the gingival crevice into the periodontal membrane. Newly formed bone from the alveolar process extending into the excavation of the dentin; the injured dentin surface is partly covered with the new cementum. Magn. 110 x.

Fig. 19. Higher magnification of figure 18; reattachment. Magn. 110 x.

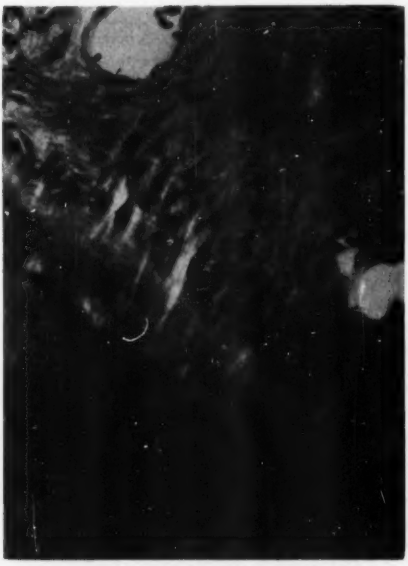


Fig. 20. The same as figure 19. Polarised light, showing collagen fibers entering the newly formed cementum. Magn. 225 x.

mentum to the natural root surface may be accounted for by as yet unknown surface characteristics of the dentin, another factor could very well be the structure of the cemental investment as a whole. This structural factor is quite clearly upset in the case of the patches of cementum on the artificial dentin surfaces of the root defects.

The organization of the bloodclot, formed after the curettage of a periodontal pocket and the gradual development of the cementum forming capacity may be assumed to proceed from the remote parts of the wound towards the cervix of the tooth. The epithelium in the meantime tends to cover the denuded surface of the cementum in order to establish a new epithelial attachment to the tooth. Whether a particular level of the root will eventually be covered with cementum (and thus be reattached) or with epithelium (and thus be lost to reattachment) is often considered to depend on the outcome of a race between the epithelium and the connective tissue.[4, 5, 6]

The comparison is scarcely valid, however, as both tissues are largely interdependent during the healing of the wound. Without the epithelial covering and some kind of epithelial attachment to the root surface, inflammation will continue to interfere with the repair of the connective tissue. On the other hand, a permanent epithelial constellation will to a large extent depend on the firmness of the connective tissue and the latters attachment to the root and the alveolar bone. Moreover, our experiments show that reattachment of the connective tissue may be absent after two or three months without the interference by epithelium. Apparently, epithelium is not the only factor preventing or delaying reattachment.

Finally, until it has been demonstrated that epithelium will never surrender cemental surface once "conquered," we should not discount this possibility.

It is highly probable that the actual relationships found in healed periodontal lesions represent the resultant of a multitude of interdependent factors rather than the outcome of a race between two tissues in the first stages of wound repair.

SUMMARY

Two types of lesions of the periodontal membrane have been made, viz., those which did and others which did not communicate with the oral cavity. In the experimental animal, the dog, reattachment was found to occur but the results suggest that the periodontal membrane may fail to reattach for months even without the presence of epithelium. Some histophysiological questions raised by the results of this investigation are discussed.

(Nic. Beetsstrast 22).

ACKNOWLEDGEMENTS

Our thanks are due to Prof. T. Jongbloed and his staff of the Physiology Department for placing at our disposal the dogs and the facilities of his laboratory. A few operations were performed in the Pharmacological Laboratory of Prof. U. G. Bijlsma, to whom we express our gratitude. The skillful assistance of Miss H. A. Bertram, who made the numerous slides, is gratefully acknowledged.

REFERENCES

1. T. D. Beckwith, Adrienne Williams and W. C. Fleming: The regeneration of rodent peridental membrane. Proc. Soc. Exp. Biol. and Med. **24**:562 (1927).

2. F. E. Beube: A study on reattachment of the supporting structures of the teeth. Journ. Periodont. **18**:55 (1947).

3. Anna Morse: Formic acid-sodium citrate decalcification and butylalcohol dehydration of teeth and bones for sectioning in paraffin. Journ. Dent. Res. **24**:143 (1945).

4. W. G. Skillen and G. R. Lundquist: An experimental study of peridental membrane reattachment in healthy and pathologic tissues. Journ. Am. Dent. Assn. **24**:175 (1937).

5. H. M. Goldman: The relationship of the epithelial attachment to the adjacent fibers of the periodontal membrane. Journ. Dent. Res. **23**:177 (1944).

6. M. Shapiro: Reattachment in periodontal disease. J. Periodontol. **24**:26 (1953).

A Quantitative Evaluation of the Effect of Copper on Alveolar Bone Loss in the Syrian Hamster*

by EMMETT R. COSTICH, D.D.S., PH.D., *Rochester, N. Y.*

COPPER deficiency has been shown to produce a bone disorder in dogs resulting in lameness or deformities of the legs.[1] Oral examination of these dogs showed the teeth to be normal with no bleeding of the gingiva or mucous membranes. Cunningham[2] has observed spontaneous fractures in sheep and cattle grazing on pastures of New Zealand that were found to have a copper deficiency in the soil. Copper has been shown to be effective in reducing salivary acid production,[3] and tooth decay in the Syrian Hamster.[4]

Because copper was effective in reducing hamster caries and because it is important in bone metabolism the following study was done to determine the effectiveness of copper in reducing alveolar crest resorption in the hamster.

METHODS

Thirty-six male and twenty-eight female hamsters were divided into four litter-mate groups, placed on a caries producing diet and given copper sulfate in their drinking water in the following amounts: Group I, 50 parts per million (ppm); Group II, 25 ppm; Group III, 10 ppm; Group IV, controls.† The water was distilled and the diet‡ and water were given *ad libitum*. The female group III became ill from undetermined causes and several died, eliminating this group from the study. At the end of 10 months the males were sacrificed and the females were sacrificed at one year. The choice of time was determined by the extent of caries development in the control animals.

The jaws were fixed in 10% neutral formalin, washed and stripped of soft tissue. To obtain a quantitative estimation of the bone loss, the height of the alveolar crest was observed and recorded on special charts (Fig. 1). From these charts the area between the cemento-enamel junction and the observed alveolar crest was traced on plastic sheets. (Eastman Kodak Kodapak #010 Frosted.) The tracing was then cut out and weighed, the weight serving as a measure of bone loss and called the periodontal score. Repeated tracings and cuttings of the same chart revealed a variation between the extremes of only 1.2%. The repeated recording of the same jaw on the special charts showed 10% difference between extremes. It was thus felt that the method of scoring alveolar crest resorption was reliable.

RESULTS

The number of animals, mean score, standard deviation and critical ratios for the animals receiving copper sulfate in the drinking water are found in Table I. It can be seen that there was a sex difference with almost twice as much bone loss in the control females as compared to the control males. In both males and the females 50 and 25 ppm of copper sulfate in

*From the Department of Dentistry, University of Rochester, School of Medicine and Dentistry and The Eastman Dental Dispensary, Rochester, New York. New address is University of Michigan School of Dentistry, Ann Arbor, Michigan.

This paper is based, in part, upon the thesis of the author submitted in partial fulfillment of the requirement for the degree of Doctor of Philosophy, June 1954.

Read by title at the 32nd General Meeting of the International Association for Dental Research, French Lick Springs, Indiana, March 19-21, 1954. (J. Dent. Res. 33:704, 1954).

†The caries study was done by Dr. John W. Hein (4).

‡Caries producing diet: 30% whole wheat flour, 30% whole powdered milk, 20% cornstarch, 15% confectionery (xxxx) sugar, 4% alfalfa, 1% NaCl.

TABLE I

Statistical Analysis of Alveolar Crest Bone Loss in Animals Receiving Copper as $CuSO_4$ in Their Drinking Water

Sex	Treatment	No. of Animals	Mean Score	S.D.
M	Control	9	1193.7	685.78
M	50ppm	10	554.9	104.60
M	25ppm	10	576.7	322.25
M	10ppm	9	975.9	514.02
F	Control	7	2366.0	919.07
F	50ppm	7	1233.1	924.17
F	25ppm	8	1122.1	867.61

CRITICAL RATIO

Control Male vs Control Female	1.65
Control Male vs 50ppm Male	2.56
Control Male vs 25ppm Male	2.46
Control Male vs 10ppm Male	0.76
Control Female vs 50ppm Female	2.30
Control Female vs 25ppm Female	3.02

$$\text{Critical Ratio} = CR = \frac{m_1 - m_2}{\sqrt{\frac{\sigma_1}{n_1} + \frac{\sigma_2}{n_2}}}$$

m = mean
n = number of degrees of freedom
σ = standard deviation
CR > 2.00 indicates significance

the drinking water produced a statistically significant reduction in the alveolar crest resorption. Although the 10 ppm males showed a slight reduction it was not considered significant.

Table II lists the animals by litters along with their caries scores, peridontal scores and weight gains. There was no relationship between periodontal or caries scores and siblings, nor was there a relationship between caries scores and periodontal scores within the experimental grouping. Periodontal scores were divided by ten and rounded off to reduce the number of digits to permit comparison with the caries scores on bar graphs. The weight gained during the experiment did show a relationship to copper concentration in the water, with the weight increasing slightly as the copper content increased to 25 ppm (Table III).

DISCUSSION

From this study it would appear that in the hamster, copper is effective in reducing alveolar bone loss as well as reducing caries. There appears to be no direct rela-

TABLE II

Litter Grouping of Periodontal Score, Caries Score, Weight Gain and Amount of Copper in the Drinking Water

Litter	Number	Sex	Amount of Cu (ppm)	Total Caries Score	Periodontal Score	Wt. gain During Experiment (grams)
A	1	M	Control	35.75	68	60
	5	M	Control	26.75	46	80
	2	M	10	26.50	82	44
	6	M	10	7.75	84	52
	3	M	25	18.00	83	78
	7	M	25	21.75	52	64
	4	M	50	4.75	68	74
B	11	F	Control	109.25	207	52
	219	M	Control	64.50	91	60
	14	M	10	10.50	65	56
	19	M	10	73.75	76	48
	15	F	25	46.25	40	60
	16	M	25	34.50	51	68
	10	M	50	34.00	54	48
	17	M	50	3.25	33	60

TABLE II *(Continued)*

Litter Grouping of Periodontal Score, Caries Score, Weight Gain and Amount of Copper in the Drinking Water

Litter	*Number*	*Sex*	*Amount of Cu (ppm)*	*Total Caries Score*	*Periodontal Score*	*Wt. gain During Experiment (grams)*
C	20	M	Control	56.75	152	60
	21	M	10	7.25	86	88
	22	M	25	11.25	54	64
	24	M	25	6.75	9	88
	23	M	50	0.00	86	92
D	30	M	Control	9.75	107	68
	32	M	25	21.25	17	48
	33	M	50	1.75	23	56
	34	M	50	23.50	45	40
E	40	M	Control	62.00	224	48
	41	M	10	9.25	92	64
	42	M	25	7.25	79	100
	43	M	50	1.75	90	60
F	50	M	Control	30.25	66	92
	52	M	10	30.25	96	68
	53	M	25	27.50	65	76
	51	F	25	22.75	205	96
	56	F	50	6.00	282	80
G	61	F	Control	58.75	188	40
	62	M	10	6.25	17	60
	64	F	50	4.25	47	60
	65	M	50	4.00	2	52
H	70	M	Control	6.25	36	80
	71	F	Control	30.75	118	44
	73	F	25	9.75	111	68
	74	M	25	4.50	0	38
	75	M	50	7.75	8	52
	76	F	50	9.75	54	?
I	82	F	Control	48.00	114	56
	80	M	Control	153.25	151	56
	81	M	10	52.75	203	72
	84	M	25	11.00	50	72
	83	F	25	5.50	36	60
	85	F	50	13.00	95	60
J	90	F	Control	27.50	189	64
	92	F	25	5.00	38	84
	96	F	25	89.00	233	96
	93	F	50	6.00	42	60
K	100	F	Control	60.50	303	66
	102	F	50	39.75	154	80
L	110	F	Control	282.00	353	32
	111	F	50	20.50	49	78
	112	F	50	13.75	76	64
M	122	F	25	54.25	88	72

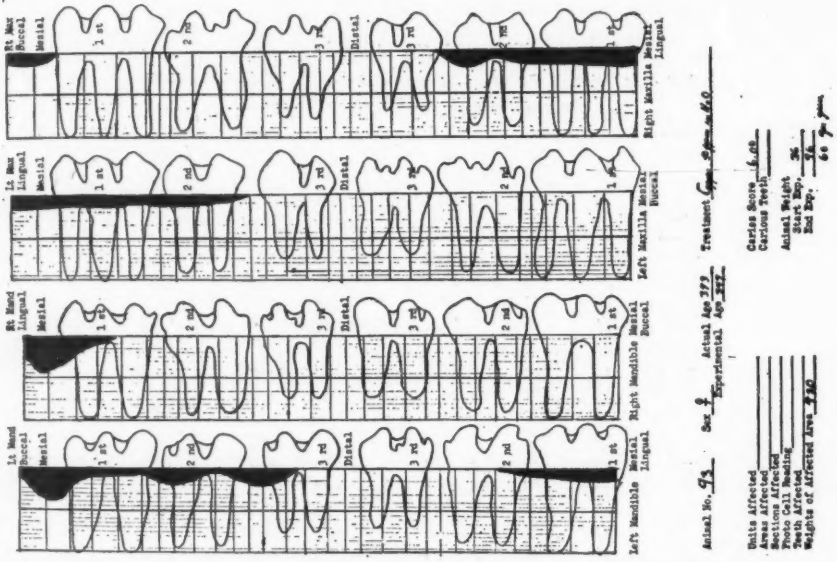


Fig. 1-A. The blackened area on the chart represents the alveolar crest resorption in a female hamster receiving 50 ppm of copper in the drinking water. Along the right margin are spaces for recording, by various methods, the bone loss observed. The age, sex, animal number, treatment, weight and, if desired for comparative purposes, the caries score obtained from a caries chart are also recorded in the spaces provided.

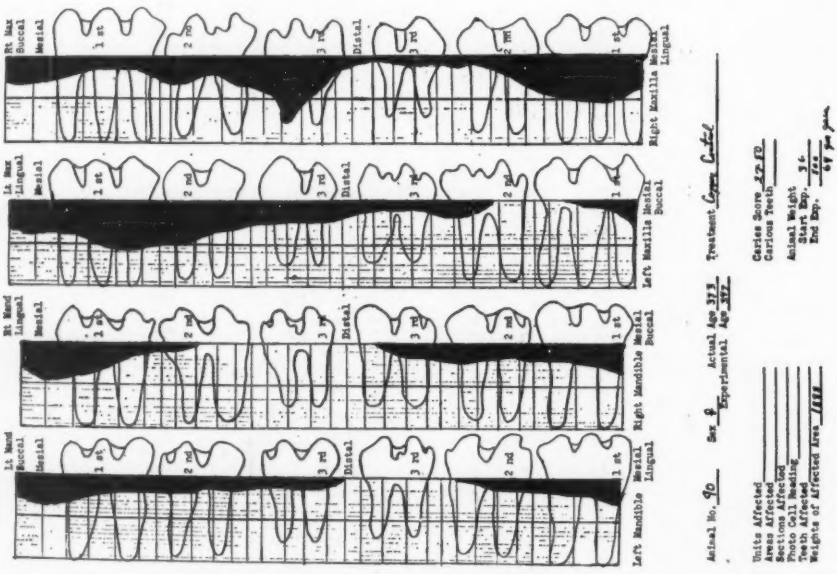


Fig. 1-B. This chart represents the findings on a litter-mate control to the animal shown in Figure 1-A. The difference in amount of alveolar crest resorption is quite noticeable.

TABLE III

Mean Weight Gain of the Hamsters Receiving Copper as $CuSO_4$ in the Drinking Water

MALE				FEMALE		
No. of Animals	*Total Weight Gain*	*Mean Weight Gain*	*Group*	*No. of Animals*	*Total Weight Gain*	*Mean Weight Gain*
9	604**	67.1	Control	7	354	50.6
9	552	61.3	10ppm	—	—	—
10	696	69.6	25ppm	8	630	78.8
10	534	53.4	50ppm	6*	404	67.3

*One animal was found dead on the day all the animals were sacrificed so no final weight could be taken due to dehydration.

**Weight in grams.

tionship between the two since the teeth and surfaces most frequently involved by caries are in areas least affected by alveolar bone loss.

The fact that the increased concentration of copper was associated with weight gain suggests a systemic influence.

Copper is known to be a strong enzyme poison and probably acted as such in the reduction of hamster tooth decay.[4] Further evidence of the action is gained from the work of Brudevold and Steadman[5] which showed no correlation between copper content of enamel and enamel solubility.

Whether the reduction in alveolar crest resorption reported in this study is due to local or systemic effects or both will require further study. The possible use of copper as a therapeutic agent for the control of caries and periodontal disease can only be determined by further investigation.

SUMMARY

1. A method for recording and measuring alveolar crest resorption in the hamster is described.

2. An evaluation of the effectiveness of copper sulfate in reducing alveolar crest resorption in the hamster in concentrations of 10, 25 and 50 ppm in the drinking water was made.

3. Copper sulfate in the concentration of 25 and 50 ppm were found to be equally effective in reducing alveolar crest resorption.

4. No relationship between caries incidence and the amount of alveolar crest resorption was found.

5. An improvement in the appearance and weight of the animals receiving copper was observed.

REFERENCES

1. Baxter, J. H. and Van Wyk, J. J.: A Bone Disorder Associated with Copper Deficiency.I.; Bull. Johns Hopkins Hosp., 93:1, 1953.

2. Cunningham, J. J.: Copper and Molybdenum in Relation to Diseases of Cattle and Sheep in New Zealand, A Symposium on Copper Metabolism; edited by W. D. McElroy, Baltimore, The Johns Hopkins Press, 1950.

3. Forbes, J. C. and Smith, J. D.: Studies on the Effect of Metallic Salts on Acid Production in Saliva; J. Dent. Res., 31:129, 1952.

4. Hein, J. W.: Effect of Copper Sulphate on Inhibition and Progression of Dental Caries in the Syrian Hamster; J. Dent. Res., 32:654, 1953.

5. Brudevold, F. and Steadman, L. T.: A Study of Copper in Human Enamel; J. Dent. Res., 34:209, 1955.

Case Report • • • •

Successful Handling of Gingival Enlargement of Complex Etiology

by EDWARD BRESSMAN. D.D.S., *Irvington, N. J.*

A FOURTEEN year old white female presented for treatment at the Periodontia clinic of New York University College of Dentistry with gingival enlargement most predominant in the upper anterior region, and to a lesser extent in the lower anterior region and the posterior regions (Figs. 1, 2). A bilateral crossbite was found in the molar regions.

Past medical history showed the usual childhood diseases, including measles, chicken pox, mumps, and whooping cough. A tonsillectomy was performed at the age of twelve. Menarche occurred at approximately eleven and one-half years. A period of five months elapsed before the second period, and then a normal pattern of thirty days was established.

Dental History. The gingival enlargement began at the age of nine. In September, 1950, the school nurse referred the patient to a hospital for treatment of this condition. For two months, "pills" and hydrogen peroxide mouth washes were employed, but the enlargement persisted. Two years later, the patient went to another hospital where a tonsillectomy was performed because the medical department of the hospital was of the opinion that a focus of infection from the tonsils existed. After the tonsillectomy, the enlargement persisted. At this time, the dental department of the same hospital suggested that the enlargement be removed surgically, but no parental consent was received.

LABORATORY FINDINGS

White Blood Count—6,200
Red Blood Count—4,520,000
Polymorphonuclear neutrophiles mature—47%
Stab.—0
Polymorphonuclear eosinophiles—6%
Polymorphonuclear basophiles—0
Lymphocytes—46%
Monocytes—1%
Hemoglobin—14.0 GMS/100 C.C. 90%
Bleeding time—1 minute 55 seconds
Coagulation time—4 minutes 20 seconds
The differential blood count showed a slight lymphocytosis.

Serum Cholesterol. 175mg. per 100 ml. of serum, which was within normal limits.

URINE ANALYSIS

Color—Amber
Reaction—6
Specific Gravity—1.025
Albumin—Negative
Sugar—Negative
Acetone—Negative
R.B.C.—Negative
W.B.C.—Negative
Casts—Negative
Crystals—Uric Acid Crystals
Epithelial Cells—Negative
Mucus—Negative

The sugar tolerance test showed a slightly flattened curve.

An evaluation of the diet revealed high carbohydrate intake with a low protein consumption, and essentially non-detergent.

Time of Specimen	Mg. %	Urine For Sugar
Fasting	93.0	Neg.
½ Hr.	115.5	Neg.
1 Hr.	121.2	Neg.
2 Hrs.	81.3	Neg.
3 Hrs.	79.8	Neg.

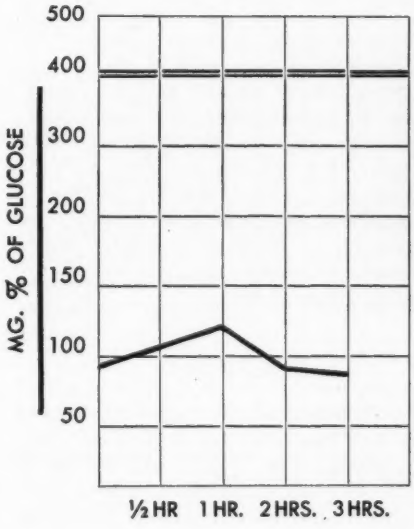


Fig. 1

Fig. 2

Fig. 3

Examination of the oral cavity showed an irregular gingival enlargement that was moderately firm, red, lobulated, and bled on palpation. No pain was present. Large deposits of supra and sub-gingival calculus were apparent. Lack of oral sanitation and a mouth-breathing habit were evident. The gingival tissues on the lingual aspect were inflamed but not enlarged. *Clinical impression*—gingival enlargement of chronic inflammatory type.

ROENTGENOGRAPHIC EXAMINATION

A tendency towards rapid alveoloclasia was revealed around the lower central incisors and the upper and lower first molars which indicated the possibility of precocious advanced alveolar atrophy. Slow alveoloclasia was present around the upper incisors and premolars.

BIOPSY REPORT

Gross: Exuberant strip of gingival tissues, canine to canine.

Microscopic: Section confirms exuberant gingival tissues described in the gross. Both roofing and subsurface tissues have participated but sheer bulk is provided by increases in deep to surface fibrils and cells. Thickening of the epithelial palisade is extremely uneven. There are several fissures and cracks where ulceration has occurred at the fundus of the fissures. Here, epithelium, once abraded, is now reparative and the tissues deep to this repair are loaded with chronic reactive cells. All looser tissue planes deeply between bundles of coarse mature collagen show the same collections of chronic cells, plasma cells predominating.

Diagnosis: Hypertrophic changes in gingival tissue. No signs of malignancy.[1]

Diagnosis—Gingival Enlargement

ETIOLOGY

Local—1. Supra and sub-gingival calculus.
2. Poor oral sanitation.
3. Non-detergent diet.
4. Mouth breathing.
5. Food impaction.

Systemic—1. Hormonal.
2. Puberty.
3. Unbalanced diet (High carbohydrate, low protein).

TREATMENT

Local — Gingivectomy with the high frequency knife was used to remove the enlargement in the region from upper canine to canine only, before other measures were instituted. The remainder of the mouth was treated by scaling and subgingival curettage and removal of prematurities of occlusion. Both the Charters and the modified Stillman methods of toothbrushing were instituted along with the use of a rubber tip for interdental stimulation. (Fig. 3 after completion of treatment.)

Systemic—Vitamin C (Ascorbic Acid) 500 mg. daily. High protein, low carbohydrate diet.

SUMMARY

A case of gingival enlargement is presented in which both local and systemic etiologic factors were evident. According to Miller,[2] a combination of factors which both lowers the tissue resistance and acts upon this weakened tissue is necessary to establish disturbances of the periodontium.

Two types of local therapy were combined effectively to obtain a result. It was felt that the large mass of tissue in the upper anterior region could be removed quickly by use of the high frequency knife, and the remainder of the mouth treated with scaling and curettage, along with adequate home care. Unless the gingival enlargement interferes with function and esthetics, it should be considered possible usually to attain its reduction without surgical intervention.

REFERENCES

1. Darlington, Charles G., M.D. Personal communication.

2. Miller, S. C., "Textbook of Periodontia." The Blakiston Company, 1950.

The Nitrogen and Lipid Content of Compact Bone from Guinea-Pig Mandible

by PHILIP PERSON, D.D.S., PH.D.,* AND GEORGE W. BURNETT, D.D.S., PH.D.,
Washington, D.C.

PROTEIN is an indispensable component of all living protoplasm, and the metabolism of any tissue is in direct proportion to the nature, amount, and activity of the living chemical matrix of protein within it.[1, 2] The metabolic responses of a bone, such as the mandible, are complex, for at any moment during its life history different areas within it vary with respect to growth, maintenance, and repair. The histology of bone is also complex, for within a single bone section may be encountered such varieties as immature bone, mature bone, bundle bone, mosaic bone, linen bone, burlap bone, etc.[3, 4] It is reasonable to propose that metabolic processes and the morphology and histology of bone are interrelated and that eventually biochemistry and physical chemistry will contribute to a fuller understanding of these phenomena and their interrelation. We may also expect that as these phenomena are explained, the protein and lipoprotein moieties of bone will be involved, since they are indispensable components of intracellular metabolic units in nearly all tissues.[5]

The relationship of the various chemical components of bone to its metabolic activity has been of particular interest to us in this laboratory, as part of our studies of the "Dynamic Equilibria of Oral Tissues".[6, 7, 8] In this article results are presented of the determination of the nitrogen and lipid content of several areas of compact mandibular bone from adult, male guinea pigs.

Department of Dental Research, Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington 12, D. C.

*Address now is Chief, Dental Research, Veterans Administration Hospital, Brooklyn 9, N. Y.

METHODS

Bone for nitrogen and lipid analysis was obtained from adult, male guinea pigs which were maintained on a diet of Purina Rabbit Chow E and water ad libitum, supplemented daily with mixed, chopped cabbage, carrots, lettuce, and kale. At the end of three months on this regimen, the animals were anaesthetized with ether and sacrificed by decapitation. The mandibles were removed and freed of muscle attachments, periosteum, and other soft tissues. The teeth were extracted, and the sockets cleansed of soft tissue. Scalpel blades and steel burs were used for cleaning. The criterion for determining complete removal of soft tissue was visual inspection, at times checked with a 6x magnifying lens. Any marrow visible to the naked eye or with the 6x lens was removed from the specimens. Each of the mandibles was then divided into six areas for sampling of compact bone as is shown in Fig. 1. Areas 1 and 2 are regions of muscle attachment; such bone is a thin, flat, compact plate, with no visible marrow content. Areas 3 and 4 are the lingual and buccal alveolar plates, respectively. Both of these samples were taken from the alveolar crest and the bone immediately inferior to it, overlying the molar roots. In the guinea pig, this bone, both labially and lingually, is essentially a single compact plate, containing little or no marrow. Area 5 is the alveolar bone overlying the roots of the lower incisor teeth on their mesial aspect; it also forms one-half the mandibular symphysis. Area 6 is the alveolar bone overlying the lateral surface of the roots of the lower incisors.

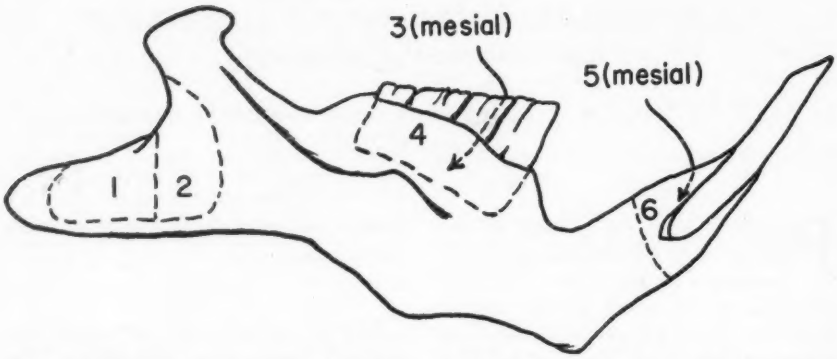


Fig. 1. Diagrammatic representation of the lateral aspect of one-half the mandible of the guinea pig, depicting the location of the areas from which samples of compact bone were taken for chemical analysis.

The bone samples prepared from the different areas were each placed in separate tared, glass-stoppered vessels and weighed. All weighings were made to the nearest tenth of a milligram, on a balance with an accuracy of ± 0.05 mg. Since the individual bone samples only weighed from 10 to 80 mg. they were each used for only one analysis. The samples were next dried to constant weight at 100°C, after which they were extracted for 4 hours with a mixture of 50 per cent ether—50 per cent ethyl alcohol (v/v), and redried to constant weight at 100°C. Four hours of extraction was found to be sufficient to remove all of the ether-alcohol extractable material from the small bone samples. The weight lost during extraction was considered to be lipid.[9] The nitrogen content of the extracted bone was then determined by a standard micro-Kjehdahl method.[10] The precision of this method is indicated by the following analysis, in triplicate, of a sample of adult rabbit femur diaphysis. The bone, prepared in the manner described above and divided into 3 portions, yielded 1.943 per cent, 1.942 per cent, and 1.947 per cent nitrogen, respectively.

RESULT AND DISCUSSION

The nitrogen content of compact mandibular bone from ten animals, together with a statistical analysis of the value, is shown in Table I. The range of the nitrogen content of the six areas was from 1.85 per cent to 4.01 per cent. The least nitrogen was found in area 3 (alveolar bone from the lingual lower molar area), with the average value of the left and right sides being 2.20 per cent. The next highest nitrogen contents were found in area 4 (alveolar bone, labial, lower molar area) and area 6 (alveolar bone, incisor lateral root surface), with the respective average values for the left and right sides being 2.74 per cent and 2.68 per cent. Area 5 (alveolar and symphyseal bone from incisor mesial root surface) had next to the highest nitrogen content, with the average of the left and right sides being 3.16 per cent. The most nitrogen was found in areas 1 and 2 (muscle attachments), with the respective averages for the left and right sides being 3.63 per cent and 3.51 per cent. Inspection of these data in Table I gives the impression that certain similarities and differences exist in the nitrogen composition of the various bone areas. To rigorously test such an impression, critical ratios were determined for the differences in their nitrogen content. A critical ratio, in this case, is the difference between the mean nitrogen content of any two areas of bone divided by its standard error. If the critical ratio is more than two the values compared are considered to vary significantly, and if it is less than two the differences are

TABLE I

The Nitrogen Content of Various Areas of Compact Mandibular Bone from Ten Guinea Pigs

Area	*% Range*	*% Mean*	*Standard Deviation*	*Coefficient of Variation*	*Standard Error of the Mean*
1 L	3.344-3.848	3.642	0.136	3.750	0.0432
1 R	3.384-4.014	3.657	0.198	5.419	0.0627
2 L	3.240-3.839	3.536	0.187	5.311	0.0594
2 R	3.066-3.835	3.490	0.221	6.343	0.0700
3 L	2.093-2.309	2.216	0.088	4.007	0.0281
3 R	1.849-2.426	2.185	0.164	7.542	0.0521
4 L	2.355-3.417	2.820	0.299	10.602	0.0945
4 R	2.278-3.059	2.667	0.274	10.303	0.0869
5 L	2.395-3.541	3.113	0.307	9.877	0.0972
5 R	3.021-3.369	3.215	0.135	4.202	0.0427
6 L	2.425-2.980	2.698	0.170	6.315	0.0538
6 R	2.358-2.892	2.662	0.166	6.258	0.0526

L—Left side of Mandible
R—Right side of Mandible

Principal Areas of Similar Nitrogen Concentration:
A. 1L, 1R, 2L, and 2R
B. 3L and 3R
C. 4L, 4R, 6L, and 6R
D. 5L and 5R.

The critical ratio was less than two when comparing the following areas:

1L vs. 1R 1R vs. 2L 3L vs. 3R 4L vs. 6R 5L vs. 6R
1L vs. 2L 1R vs. 2R 4L vs. 4R 4R vs. 6L 6L vs. 6R
1L vs. 2R 2L vs. 2R 4L vs. 6L 4R vs. 6R

The critical ratios for all other comparisons was more than two.

TABLE II

Protein Content of Compact Mandibular Bone from the Guinea Pig (% N_2 x 5.55)

Area Description	*Mean % N_2*	*% Protein*
1. Muscle Attachment	3.63	20.15
2. Muscle Attachment	3.51	19.48
3. Lingual Alveolar Plate (Molar)	2.20	12.21
4. Labial Alveolar Plate (Molar)	2.74	15.21
5. Mesial Alveolar Surface (Incisor)	3.16	17.54
6. Lateral Alveolar Surface (Incisor)	2.68	14.87

TABLE III

The Lipid Content of Various Areas of Compact Mandibular Bone from Six Guinea Pigs

Area	*% Range*	*% Mean*	*Standard Deviation*	*Coefficient of Variation*	*Standard Error of the Mean*
1 L	0.568-1.612	0.878	0.410	46.77	0.1677
1 R	0.264-2.702	0.925	0.919	99.35	0.4596
2 L	0.327-1.742	0.854	0.547	64.13	0.2236
2 R	0.286-0.961	0.660	0.292	44.28	0.1193
3 L	0.575-1.473	1.047	0.295	28.23	0.1207
3 R	0.545-2.114	0.969	0.569	58.77	0.2325
4 L	0.588-1.709	1.142	0.530	46.44	0.2165
4 R	0.116-2.127	1.074	0.832	77.47	0.3397
5 L	0.502-2.00	0.854	0.578	67.70	0.2360
5 R	0.409-1.272	0.869	0.342	39.43	0.1399
6 L	0.307-1.047	0.575	0.281	48.86	0.1147
6 R	0.411-1.020	0.724	0.259	35.85	0.1060

L—Left side of Mandible
R—Right side of Mandible

The critical ratio for all possible comparisons was less than two, except for 2R versus 3L, 3L versus 6L, 3L versus 6R, 4L versus 6L. It is apparent, however, that these results cannot be interpreted as indicating any patterns of organization into groups, with respect to lipid content.

likely to have occurred by chance.[11] The critical ratios of the 66 possible comparisons indicated that 4 significantly different nitrogen containing areas of bone were present in the mandible. On the other hand, most of the critical ratios that were less than two were for comparisons of right and left sides, none of which were more than two. Thus, it is interesting to note that there was no apparent difference in total nitrogen content between the right or left halves of a given area.

The nitrogen found in the mandibular bone samples is derived from osteocytes, Haversian and Volkmann canals, intercellular fluids, calcified matrix, and Sharpey's fibers. In these structures the protein present in greatest amount is collagen. Hence, we may estimate the protein content of the bone areas, calculated as collagen, in the following manner. Collagen contains approximately 18 per cent nitrogen; therefore, if we multiply the nitrogen analysis values by a conversion factor of 100 divided by 18, or 5.55, we will obtain the approximate protein content of the areas in terms of collagen. The results of such calculations are given in Table II.

The lipid content of compact mandibular bone from the different areas is given in Table III, representing complete analyses for six animals. The lipid content ranged from 0.12 per cent to 2.70 per cent. Unlike nitrogen, there was no consistent distribution of the ether-alcohol extractable lipids in the various regions. This impression is also borne out by the results of the statistical analysis of the data in which the critical ratios of only four of 66 possible comparisons were more than two. The only conclusion that can be made concerning total lipid content of the various regions

of mandibular bone is that it is extremely variable and unpredictable. This is indeed an interesting observation, since other compact bone, such as that of the femur diaphysis from the same animal may contain no detectable lipid. It has also been reported, using a slightly different method of analysis, that there is no lipid in the compact bone from ox femur diaphysis.[12]

SUMMARY

The nitrogen content of six different areas of compact bone from the mandibles of adult guinea pigs was found to range from 1.85 per cent to 4.01 per cent. The lowest nitrogen, and hence protein content, was found in alveolar bone, while the highest nitrogen content was found in muscle attachment areas. The total nitrogen content of similar areas of bone from both sides of the mandible did not differ. The total lipid content of the same areas showed extreme variation in the same and in different areas. Compact bone from femur diaphysis of the same animals contained no detectable lipids.

BIBLIOGRAPHY

1. Haurowitz, F.: Chemistry and Biology of the Proteins. New York, 1950. Academic Press. p. 1.

2. Allison, J. B.: The Metabolism of Proteins and Amino Acids. Ann. Rev. Biochem., 17:275, 1948.

3. Weinmann, J. P., and Sicher, H.: Bone and Bones. St. Louis, 1947. C. V. Mosby and Co. Chaps. 1, 2, and 3.

4. Ham, A. W.: Histology. Philadelphia, 1950. J. B. Lippincott Co. Chap. 16.

5. Bensley, R. R., and Hoerr, N.: The Preparation and Properties of Mitochondria. Anat. Rec., 60:449, 1934.

6. Person, P.: Dynamic Equilibria of Oral Tissues. I. Theoretical Considerations. J. Periodont. 26:7, 1955.

7. Person, P., and Burnett, G. W.: Dynamic Equilibria of Oral Tissues. II. Cytochrome Oxidase and Succinoxidase Activity of Oral Tissues. In Press, J. Periodont.

8. Person, P., and Burnett, George W.: Dynamic Equilibria of Oral Tissues. III. The Effect of Acute Starvation Upon Cytochrome Oxidase and Succinoxidase Activity of Oral Tissues. In Press, J. Periodont.

9. Hsaio, S. C.: A Simple Micromethod for Rapid Extraction of Lipids. Science 107:24, 1948.

10. Neiderl, J., and Neiderl, V.: Micromethods of Quantitative Organic Elementary Analysis. New York, 1938. J. Wiley & Son, p. 51.

11. Hill, A. B.: Principles of Medical Statistics. London, 1948. The Lancet, Limited.

12. Eastoe, J. E., and Eastoe, B.: The Organic Constituents of Mammalian Compact Bone. Biochem. Jour., 57:453, 1954.

WESTERN SOCIETY OF PERIODONTOLOGY

The Western Society of Periodontology will hold its annual meeting at the Sahara Hotel, Las Vegas, Nevada, on March 16, 17, 18, 1956. The program will feature Dr. Henry Goldman.

For further information write Dr. Robert L. Reeves, President of the Society, at 718 Wilshire Blvd., Santa Monica, California.

Editorial • • • •

Is Dental Practice Changing?

A year or so ago the American Dental Association published a series of studies entitled "The Survey of Need for Dental Care"[1] which warrants careful study. This survey was made of individuals who had sought dentists' offices and should not be considered as a survey of the general population. Findings of the survey included:

1. The male dental patient between the ages of 15 and 19 needed an average of six fillings while the female patient in this group needed 5.4. In the 20-24 year age group the figures were 4.4 and 4.6 fillings needed.
2. The average patient in 1952 needed two extractions if a male and 1.6 extractions if a female.
3. 14.3% of the male patients and 11.3% of the female patients required a full upper denture. 11% of the males and 8.2% of the females required a complete lower denture. In the age group of 55-64 approximately one-third of the males required an upper denture and one-fourth of them required a lower denture.

These figures compare favorably with those from a study made in 1940.[2] At that time the average male patient needed 4.3 fillings as compared with 2.9 fillings in 1952 and needed 2.7 extractions in 1940 as compared with 2.0 in 1952. The average female patient required 4.4 fillings in 1940 as compared with 3.1 in 1952. She required two extractions in 1940 as compared with 1.6 in 1952. In 1940 the average male visited the dentist once in 2-½ years and in 1952 he saw his dentist after only 1.9 years. For the female the average frequency of visits was 2.2 years in 1940 and 1.6 years in 1952. These two studies would seem to indicate that there is a slowly improving pattern of dental care over this twelve year period.

---

1. (a) A Survey of needs for dental care I. Methodology and composition of sample. J.A.D.A. vol. 45, p. 706, December 1952.
   (b) Survey of needs for dental care II. Dental needs according to age and sex of patients. J.A.D.A. vol. 46, p. 200, February 1953.
   (c) Survey of needs for dental care III. Dental needs according to length of time since last visit to a dentist. J.A.D.A. vol. 46, p. 562, May 1953.
   (d) Survey of needs for dental care IV. Dental needs according to region of the country. J.A.D.A. vol. 47, p. 206, August 1953.
   (e) Survey of needs for dental care V. Dental needs according to income and occupation. J.A.D.A. vol. 47, p. 340, September 1953.
   (f) Survey of needs for dental care VI. Summary and comparison with 1940 survey. J.A.D.A. vol. 47, p. 572, November 1953.
2. Study of the dental needs of adults in the United States. Economics Committee of the A.D.A. 1940. Cited in 1(f).

It will be important to observe the effect of wide-spread fluoridation of communal water supplies on the practice of dentistry in the next decade. It is entirely probable that the number of restorations needed will diminish and the speed of development of dental caries will be reduced. As a result, it is possible that operative procedures may be modified. However, apparently no more than 40% of the public visit a dentist each year. From all evidence that we have at the moment, it is possible that there will be little change in the demand for dentures, since up to the present time no method has been found of preventing periodontal disease which, as is well known, causes the loss of most teeth for the adult. No method of prevention, that is, except that which must be given by a dentist.

Nevertheless, the pattern of dental needs of the public may change in the next generation and with changes dental education and dental practice must change also. It behooves every dental practitioner and dental teacher to keep informed of the changes in dental needs, as well as advances in dental technics and dental materials.

It also behooves all dentists to keep informed of developments in dental research, and to support research in every possible way. The editor readily admits that certain of the articles in this issue of the Journal are not applicable to private practice of dentistry, and perhaps never will be. However, expansion of some of the studies reported here could conceivably lead to clinical applications of much importance. This is the way of progress.

Announcements • • • •

UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF DENTISTRY

The School of Dentistry of the University of Southern California is presenting a post-graduate course in Periodontics this fall. The course is under the direction of Dr. Robert L. Reeves with instruction also by Arnold A. Ariaudo, Nathan Friedman, Perry A. Ratcliff, and Sylvan Schireson. Tuition is $200.00 per semester. Enrollment is limited to ten.

For further information write Dr. Reeves at The University of Southern California School of Dentistry, 925 West Thirty-Fourth Street, Los Angeles 7, California.

INDIANA UNIVERSITY SCHOOL OF DENTISTRY

Indiana University School of Dentistry is presenting a three-day symposium on periodontology November 29, 30 and 31. Emphasis will be placed upon diagnosis and treatment of periodontal diseases. Lectures and demonstrations will be given by Drs. Henry Swenson, Grant Van Huysen, David Mitchell, William Shafer, John Johnston, Maury Massler, Lewis Spear and Maynard K. Hine.

There will be a $50 fee and the enrollment will be limited.

For details write the Dean, 1121 West Michigan Street, Indianapolis 2, Indiana.

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY
POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Samuel Charles Miller and Staff:

No. 124. REFRESHER COURSE IN PERIODONTIA AND ORAL MEDICINE

Ten sessions, Wednesdays, 4:00 to 6:00 P.M.
February 29, 1956 to May 10, 1956.
Tuition and books. $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occlusion is performed by the student on casts. Patient demonstrations include equilibration of occlusion, conservative and radical therapy, home care, etc.

No. 123. PERIODONTIA AND ORAL MEDICINE IN THE SPANISH LANGUAGE

Four weeks, Monday through Friday, 9:00 A.M. to 4:00 P.M.
April 2 to April 27, 1956 inclusive.
Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

No. 122. COMPREHENSIVE PREPARATION IN PERIODONTIA AND ORAL MEDICINE

No. 122 A. One academic year, full time. September 19, 1955 to May 25, 1956.
Tuition $1,010.00. Instruments and books approximately $135.00.

No. 122 B. Two academic years, full time. September 19, 1955 to May 24, 1957.
Tuition $1,770.00 (two years). Instruments and books approximately $231.00.

No. 122 C. Two academic years, half time. September 19, 1955 to May 24, 1957. (Two and one half days weekly.)
Tuition $1,010.00 (two years). Instruments and books approximately $135.00.

No. 122 D. Four academic years, half time. September 19, 1955 to May 29, 1959. (Two and one half days weekly.)
Tuition $1,770.00 (four years). Instruments and books approximately $231.00.

These courses are designed for comprehensive preparation in Periodontia and Oral Medicine and are of particular significance for certification towards specialization. All didactic and clinical phases are covered with the objective of developing expert proficiency.

Further information concerning the courses listed above can be obtained by writing to the Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, New York.

BETH ISRAEL HOSPITAL PERIODONTIA COURSES — SPRING 1956

The Dental Department of the Beth Israel Hospital in Boston announces two postgraduate courses in Periodontia to be given in the Spring of 1956.

A 5-day course in Occlusal Adjustment will be given from March 7 to 11. This course will include background material on the anatomy, histology and physiology of the periodontium and dental apparatus. Diagnostic methods and treatment procedures for occlusal adjustment will be thoroughly discussed and demonstrated in the clinic. Participants will be limited to twelve. The tuition fee is $200.00.

A two week seminar in Periodontia is scheduled for May 7 to May 18. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations and clinics. The tuition fee is $350.00.

Co-Directors of the Courses: Henry M. Goldman, D.M.D. and Bernard Chaikin, D.M.D.

For further information, write to: Coordinator of Education Programs, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Massachusetts.

PHILADELPHIA SECTION OF INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

The Philadelphia Section of the International Association for Dental Research is planning a comprehensive periodontal program at the University of Pennsylvania Dental School on Sunday, November 6, 1955 at 9:30 a.m. This scientific program will be presented in conjunction with the Boston, New York, Baltimore, Pittsburgh and Washington sections of the I.A.D.R.

All members of the dental profession and its allied sciences are invited.

THE UNIVERSITY OF ALABAMA SCHOOL OF DENTISTRY DIVISION OF POSTGRADUATE EDUCATION

A three-day refresher course in The Theory and Practice of Periodontics, December 3, 4, and 5, 1955, under the direction of Balint Orban, M.D., D.D.S., Professor and Chairman of the Department of Periodontics, Loyola University, Chicago College of Dental Surgery, and Director of the Colorado Dental Foundation, Colorado Springs, Colorado.

Doctor Orban was graduated from the Medical School of the University of Vienna and the School of Dentistry of Northwestern University, and has limited his practice and teaching to the specialized field of Periodontics. A world renowned periodontist, Doctor Orban was recently awarded the "Miller Prize" by the Federation Dentaire Internationale. He is the author of the textbook Oral Histology and Embryology.

The course will consist of lectures, discussions, and clinical demonstrations regarding the normal gingiva, classification and diagnosis of periodontal diseases, the epithelial attachment and gingival sulcus, the development and elimination of the periodontal pocket, the biologic considerations of the occlusal problem, tissue changes in occlusal trauma, and systemic aspects of periodontal diseases. Special emphasis will be given to the occlusion and its relation to periodontal disease.

The tuition fee for the course is $85.00 and the class enrollment will be limited to sixteen (16).

Please write to the University of Alabama School of Dentistry, Director of Postgraduate Education, University of Alabama School of Dentistry, Birmingham 3, Alabama.

NEW ROSTER OF DIPLOMATES OF AMERICAN BOARD OF PERIODONTOLOGY BEING PREPARED

A complete list of the diplomates of the American Board of Periodontology is now being prepared by Dr. Harold Leonard, Secretary of the Board, and will be available about Nov. 15. On the next page is a list of those who have become diplomates in 1955.

DIPLOMATES OF THE AMERICAN BOARD OF PERIODONTOLOGY

*Algus, Seymour, B.A., D.D.S. (1946)	1 Hanson Place, Brooklyn 17, N. Y.	Certified	1955
*Ball, Edward Lee, Jr., D.D.S. (1944)	3268 Jefferson Ave., Cincinnati 20, Ohio	"	1955
†Bradin, Morris, D.D.S. (1937)	407 Cooper St., Camden 2, N. J.	"	1955
*Clark, James William, D.D.S. (1947)	303 W. 8th St., Texarkana, Ark.	"	1955
–Flaschner, Abraham Mark, D.M.D. (1918)	139 Sherman Rd., Chestnut Hill 67, Mass.	"	1955
*Harris, Irving Michael, D.D.S. (1937)	555 Cedar Lane, Teaneck, N. J.	"	1955
xHiatt, William Henry, B.A., D.D.S. (1945)	Cody Dental Clinic, 16th Ave. at Humboldt St., Denver, Colo.	"	1955
–Hine, Maynard K., D.D.S., M.S. (1930)	1121 W. Michigan St., Indianapolis 2, Ind.	"	1955
†Ivancie, Gerald P., D.D.S., M.S. (1947)	Univ. of Iowa College of Dentistry, Iowa City, Iowa	"	1955
†Kollar, John Anton, Jr., D.D.S. (1942)	30 N. Michigan Ave., Chicago 2, Illinois	"	1955
*Moore, Robert Lansing, B.S., D.D.S. (1946)	28 Eighth St., N. E., Atlanta, Ga.	"	1955
†Nachazel, Delbert Peter, D.D.S. (1928)	425 E. Wisconsin Ave., Milwaukee 2, Wisconsin	"	1955
†Ogilvie, Alfred L., D.D.S., M.S. (1944)	Univ. of Washington School of Dentistry, Seattle 5, Washington	"	1955
*Reeves, Robert Lloyd, D.D.S. (1945)	718 Wilshire Blvd., Santa Monica, Calif.	"	1955
*Schreiber, Harold Richard, A.B., D.D.S. (1947)	3720 Washington Blvd., St. Louis, Mo.	"	1955
†Simring, Marvin, B.A., D.D.S. (1944)	2566 Ocean Ave., Brooklyn 29, N. Y.	"	1955
*Stahl, S. Sigmund, D.D.S., M.S. (1947)	5 East 57th St., New York 22, New York	"	1955
*Sumner, Charles Ferris, III, D.D.S. (1947)	Capt. USA Dental Service, Brook Army Hospital, Fort Sam Houston, Texas	"	1955
†Wachtel, Nathan, B.A., D.D.S. (1945)	480 Bedford St., Stamford, Conn. and 12 E. 86th St., New York, N. Y.	"	1955
*Ward, Howard L., B.A., D.D.S. (1949)	The Medical Center, 15 Bond St., Great Neck, N. Y.	"	1955
*Wiebusch, F. B., B.B.A., D.D.S. (1947)	Medical College of Virginia Dental School Richmond, Va.	"	1955

Interpretation of symbols:
* — practice limited to periodontia
† — practice partially limited to periodontia
x — general practice
– — not practicing
d — deceased
() — date of graduation from dental school

PERIODONTOLOGY COURSES AT COLUMBIA UNIVERSITY

Four postgraduate courses in Periodontology are offered at Columbia University, Scnool of Dental and Oral Surgery of the Faculty of Medicine.

A full-time program covering a period of two academic years and a half-time program covering a period of four academic years lead to a Certificate of Training in Periodontology. Correlation of the fundamental sciences to clinical practice is emphasized.

Of the two part-time courses, one course will be given one full day a week from September, 1955 to May, 1956; the second course will be given one half day each week from September, 1955 to May, 1956.

For further information write to Postgraduate Division, School of Dental and Oral Surgery, 630 West 168th Street, New York 32, N. Y.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE
DIVISION OF GRADUATE AND POSTGRADUATE STUDIES
BOSTON, MASSACHUSETTS, U. S. A.

ANUNCIO ESPECIAL
CURSO PRACTICO EN PERIODONCIA PARA POST-GRADUADOS

Presentado por el Profesor Irving Glickman en combinacion con los siguientes colegas: Dr. Fermin A. Carranza, Jr., Dr. Javier Restrepo, Dr. Aurelio Echeverri, Dra. Violeta A. Glickman.

Este curso sera dictado en español. Su duración será de dos semanas (de 9 a.m. a 5 p.m. seis dias semanales) iniciandose el 6 de Febrero, 1956 y terminandose el 18 de Febrero. El programa esta organizado en la forma siguiente: la mitad de cada dia sera dedicado a conferencias y seminarios y la otra mitad al tratamiento de pacientes por los participantes en la clinica de Post-Graduados.

Este curso esta abierto para dentistas de habla española graduados en Facultades Odontologicas reconocidas en sus respectivos paises.

La matricula para dicho curso es de $200. dollares. El costo aproximado de los instrumentos es de $75. dollares.

Para mayor detalles y para registrarse dirigirse al Director of Graduate and Post-Graduate Studies, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Mass., U.S.A.

72886